THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
AGRICULTURAL RESEARCH ADMINISTRATION
UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 199

WHEAT LEAF RUST STUDIES AT SAINT PAUL, MINNESOTA

Supplement 199-209

March 30, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

WHEAT LEAF RUST STUDIES AT SAINT PAUL, MINNESOTA [1]

M. N. Levine [2], E. R. Ausemus [3], and E. C. Stakman [4]

Plant Disease Reporter
Supplement 199 March 30, 1951

This report is presented in response to a recommendation adopted at the Eighth Hard Red Spring Wheat Conference, held at Saint Paul, Minnesota, on February 3-4, 1948. Three lines of investigations are summarized, viz.: (1) The occurrence and distribution of physiologic races isolated from wheat leaf rust specimens, collected in the Upper Midwest area of the United States during the 25-year period, 1925-1949, inclusive; (2) the seedling reaction of more than a hundred varieties of wheat to different physiologic races of Puccinia rubigo-vera tritici, tested under greenhouse conditions; and (3) the comparative reaction of some six dozen wheat varieties, grown in field plots at University Farm during one or more of the 10 years from 1938 to 1947, inclusive. No field notes were taken in 1948 and 1949, because of the virtual absence of leaf rust in the plots.

PHYSIOLOGIC RACES OF PUCCINIA RUBIGO-VERA TRITICI IN THE UPPER MIDWEST

Identification of wheat leaf rust races at St. Paul was started in 1925, the year leaf rust readings were begun in the cooperative uniform wheat nurseries maintained in different parts of the country. The number of isolates identified reached a substantial figure only during the past decade. As recorded in Table 1, a total of 1008 isolates were identified during the quarter century under review, averaging slightly over 40 identifications a year, but the average during the last quinquennium was somewhat in excess of 100 identifications per year. Of the four States involved in this study, Minnesota provided 59.2 percent of the isolates; North Dakota, 27.0 percent; South Dakota, 10.6 percent; and Montana, only 3.2 percent. Many persons, too numerous to mention by name, cooperated in the collection of uredial material in various parts of the area.

The isolates identified grouped themselves into 48 different physiologic races. The racial composition varied from year to year, to some extent, and from quinquennium to quinquennium, to a greater extent. Thus, while race 9 was predominant in its frequency of occurrence during the first three quinquennia, race 128 assumed the predominant position during the fourth quinquennium, but lost it to race 126 in the fifth. The five most common races during the entire quarter century were in the order listed: race 9 (10.7 percent), race 128 (10.4 percent), race 126 (8.8 percent), race 5 (8.7 percent), and race 15 (6.8 percent). Most common among the 44 races isolated from the Minnesota collections were race 128 (12.4 percent), race 126 (9.9 per-

---

[1] Joint contribution from the Division of Agronomy and Plant Genetics and the Division of Plant Pathology and Botany, Department of Agriculture, University of Minnesota, in cooperation with the Division of Cereal Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, United States Department of Agriculture. Published as Paper No. 2613 of the Scientific Journal Series of the Minnesota State Agricultural Experiment Station.

[2] Pathologist, Cereal Rust Investigations, United States Department of Agriculture, collaborating with the Division of Plant Pathology and Botany at the University of Minnesota.

[3] Agronomist, Spring Wheat Investigations, United States Department of Agriculture, collaborating with the Division of Agronomy and Plant Genetics at the University of Minnesota.

[4] Chief, Division of Plant Pathology and Botany, University of Minnesota, and Agent in Charge of Rust Surveys, Division of Plant Disease Control, Bureau of Entomology and Plant Quarantine, Agricultural Research Administration, United States Department of Agriculture.

Table 1. Physiologic races of Puccinia rubigo-vera tritici isolated from Upper Midwest

Physiologic races identified	Periodic and geographic distribution of physiologic races identified: Quinquennial periods									
	1925-1929		1930-1934		1935-1939		1940-1944		1945-1949	
	No.	%	No.	%	No.	%	No.	%	No.	%
1	-	--	2	4.9	-	--	5	1.4	22	4.2
2	-	--	-	--	-	--	9	2.5	6	1.1
3	4	10.8	1	2.4	-	--	-	--	12	2.3
4	-	--	-	--	1	2.0	-	--	3	.6
5	1	2.7	-	--	2	4.1	16	4.5	69	13.1
6	-	--	-	--	2	4.1	11	3.1	4	.8
7	-	--	-	--	-	--	6	1.7	10	1.9
9	19	51.4	15	36.6	13	26.5	17	4.8	44	8.3
10	2	5.4	-	--	1	2.0	1	.3	-	--
11	-	--	-	--	3	6.1	1	.3	4	.8
12	-	--	-	--	-	--	1	.3	1	.2
13	3	8.1	5	12.2	2	4.1	4	1.1	2	.4
14	-	--	-	--	-	--	2	.6	-	--
15	-	--	8	19.5	5	10.2	17	4.8	39	7.4
17	-	--	-	--	-	--	10	2.8	9	1.7
18	-	--	-	--	-	--	4	1.1	-	--
19	-	--	2	4.9	2	4.1	8	2.3	2	.4
20	-	--	-	--	-	--	14	4.0	-	--
21	-	--	-	--	-	--	5	1.4	2	.4
28	-	--	-	--	4	8.2	13	3.7	22	4.2
29	-	--	-	--	-	--	1	.3	-	--
31	2	5.4	4	9.8	1	2.0	11	3.1	9	1.7
32	3	8.1	-	--	-	--	-	--	-	--
33	1	2.7	-	--	1	2.0	5	1.4	7	1.3
34	1	2.7	-	--	-	--	-	--	-	--
35	-	--	-	--	-	--	33	9.3	32	6.1
37	-	--	1	2.4	1	2.0	4	1.1	17	3.2
40	-	--	-	--	-	--	1	.3	6	1.1
42	-	--	-	--	-	--	1	.3	-	--
43	-	--	-	--	-	--	1	.3	-	--
44	-	--	1	2.4	1	2.0	5	1.4	8	1.5
46	-	--	-	--	1	2.0	-	--	-	--
49	-	--	-	--	-	--	35	9.9	2	.4
50	-	--	2	4.9	1	2.0	1	.3	11	2.1
52	1	2.7	-	--	-	--	12	3.4	22	4.2
56	-	--	-	--	-	--	2	.6	-	--
58	-	--	-	--	1	2.0	-	--	2	.4
60	-	--	-	--	1	2.0	-	--	-	--
61	-	--	-	--	-	--	3	.8	-	--
64	-	--	-	--	1	2.0	4	1.1	2	.4
77	-	--	-	--	-	--	12	3.4	12	2.3
80	-	--	-	--	-	--	1	.3	-	--
90	-	--	-	--	1	2.0	1	.3	1	.2
91	-	--	-	--	-	--	4	1.1	6	1.1
93	-	--	-	--	2	4.1	-	--	8	1.5
105	-	--	-	--	2	4.1	6	1.7	4	.8
126	-	--	-	--	-	--	15	4.2	74	14.0
128	-	--	-	--	-	--	52	14.7	53	10.1
Isolates	37	100.0	41	100.0	49	99.6	354	100.0	527	100.2
Races	10		10		22		40		34	

collections obtained during 1925-1949.

Physiologic races identified	Total number of isolates	Percent of grand total	Periodic and geographic distribution of physiologic races identified: Individual States: Minnesota		Montana		North Dakota		South Dakota	
			No.	%	No.	%	No.	%	No.	%
1	29	2.9	15	2.5	6	18.8	4	1.5	4	3.7
2	15	1.5	4	.7	-	--	4	1.5	7	6.5
3	17	1.7	11	1.8	-	--	5	1.8	1	.9
4	4	.4	3	.5	1	3.1	-	--	-	--
5	88	8.7	56	9.4	2	6.2	25	9.2	5	4.7
6	17	1.7	13	2.2	-	--	3	1.1	1	.9
7	16	1.6	9	1.5	3	9.4	4	1.5	-	--
9	108	10.7	39	6.5	6	18.8	44	16.2	19	17.8
10	4	.4	1	.2	-	--	2	.7	1	.9
11	8	.8	4	.7	-	--	4	1.5	-	--
12	2	.2	1	.2	-	--	1	.4	-	--
13	16	1.6	6	1.0	3	9.4	7	2.6	-	--
14	2	.2	2	.3	-	--	-	--	-	--
15	69	6.8	34	5.7	2	6.2	27	9.9	6	5.6
17	19	1.9	12	2.0	1	3.1	4	1.5	2	1.9
18	4	.4	4	.7	-	--	-	--	-	--
19	14	1.4	5	.8	-	--	7	2.6	2	1.9
20	14	1.4	11	1.8	-	--	3	1.1	-	--
21	7	.7	2	.3	-	--	2	.7	3	2.8
28	39	3.9	30	5.0	-	--	4	1.5	5	4.7
29	1	.1	1	.2	-	--	-	--	-	--
31	27	2.7	11	1.8	2	6.2	8	2.9	6	5.6
32	3	.3	-	--	-	--	1	.4	2	1.9
33	14	1.4	4	.7	2	6.2	5	1.8	3	2.8
34	1	.1	-	--	-	--	1	.4	-	--
35	65	6.4	52	8.7	-	--	10	3.7	3	2.8
37	23	2.3	4	.7	-	--	14	5.1	5	4.7
40	7	.7	5	.8	-	--	1	.4	1	.9
42	1	.1	1	.2	-	--	-	--	-	--
43	1	.1	1	.2	-	--	-	--	-	--
44	15	1.5	5	.8	-	--	9	3.3	1	.9
46	1	.1	-	--	-	--	1	.4	-	--
49	37	3.7	35	5.9	-	--	2	.7	-	--
50	15	1.5	4	.7	1	3.1	10	3.7	-	--
52	35	3.5	24	4.0	-	--	8	2.9	3	2.8
56	2	.2	2	.3	-	--	-	--	-	--
58	3	.3	3	.5	-	--	-	--	-	--
60	1	.1	1	.2	-	--	-	--	-	--
61	3	.3	2	.3	-	--	-	--	1	.9
64	7	.7	3	.5	-	--	3	1.1	1	.9
77	24	2.4	21	3.5	-	--	3	1.1	-	--
80	1	.1	-	--	-	--	-	--	1	.9
90	3	.3	2	.3	-	--	1	.4	-	--
91	10	1.0	8	1.3	-	--	1	.4	1	.9
93	10	1.0	7	1.2	1	3.1	1	.4	1	.9
105	12	1.2	6	1.0	-	--	5	1.8	1	.9
126	89	8.8	59	9.9	-	--	18	6.6	12	11.2
128	105	10.4	74	12.4	2	6.2	20	7.4	9	8.4
Isolates	1008		597	59.2	32	3.2	272	27.0	107	10.6
Races	48		44		13		37		28	

cent), and race 5 (9.4 percent). The three most common among the 37 North Dakota races were race 9 (16.2 percent), race 15 (9.9 percent), and race 5 (9.2 percent). The three most common among the 28 South Dakota races were race 9 (17.8 percent), race 126 (11.2 percent), and race 128 (8.4 percent). Outstanding among the 13 Montana races were races 1 and 9, each constituting 18.8 percent of the State's total.

SEEDLING REACTION TO DIFFERENT PHYSIOLOGIC RACES OF WHEAT LEAF RUST

Some 200 standard varieties, hybrid selections, and foreign introductions of common wheats, durums, emmers, and einkorn were tested in the seedling stage under greenhouse conditions for their reaction to different physiologic races of leaf rust during the 12-year period, 1939-1950. The preponderant majority of races used had been isolated from collections made in the Upper Midwest area. Table 2 contains the reaction record of 113 lines, none of which had been subjected to fewer than 7 physiologic races, while several of them were tested with as many as 52 different races. All of the 48 Upper Midwest races were used in the test at one time or another. Many of the lines studied were tested to more than a single strain of a given race but, for the most part, the reaction was similar, if not altogether identical. The deviations will be pointed out later. In Table 2, the dominant reaction was recorded as either resistant or susceptible. The infection types, recorded in the original readings, were segregated into the two reaction classes in the following manner: (a) the infection types 0, 1, and 2 were incorporated into the resistant class, in which case 0 = practical immunity (no visible uredia, necrotic lesions or flecks often present), 1 = extreme resistance (minute uredia clearly visible, pustules embedded in well defined necrotic areas), 2 = moderate resistance (small uredia in abundance, necrotic lesions clearly defined but less extensive); (b) the infection types X, 3, and 4 were embodied in the susceptible class, in which case X = variable mesothesis (conglomeration of uredia of diverse size and type tending to integrate), 3 = moderate susceptibility (uredia of medium size, slight chlorosis usually accompanying infection centers), 4 = extreme susceptibility (predominantly large uredia, chlorosis rare, necrosis absent).

Among the 52 leaf rust races, used in the seedling reaction study, were eight races that consisted of more than one biotype. These were races: 1, 5, 9, 11, 15, 28, 93, and 126. Indistinguishable in their parasitic behavior on the customary differential hosts, the biotypes of each of the aforementioned races could be distinguished readily by the difference in type of infection produced on one or more additional wheat varieties as specified below. Thus, Frontana, ordinarily highly resistant to race 1, was moderately susceptible to a strain of race 1 in 1946. Most frequently, Cadet, Centenario, Kubanka, Pilot, Red Egyptian, Regent, Renown, and Rival are very resistant to race 5, but, occasionally, a biotype of race 5 produces normal infection on all of these varieties. Cincana, Maroqanith, and Pentad are resistant to some biotypes of race 5 as often as they are susceptible to other biotypes of this race. Khapli, on the other hand, usually susceptible to race 5, is resistant to an occasional strain of this race. Frontana and Red Egyptian most often are very resistant to race 9, but an occasional strain of this race attacks these two varieties rather severely. Just the opposite is true of Premier. As a rule, Lee, Frontana, Gaza, and Premier are highly resistant to race 11, but at least one strain of race 11 produces moderate to severe infections on these four varieties. In most trials, Frontana, Red Egyptian, and Regent are quite resistant to race 15, and only occasionally does a biotype of this race produce normal infection. The reverse is true of Premier. Hope, Kubanka, and Pentad are most often quite susceptible to race 28, but an occasional biotype of this race is incapable of producing normal infection on these three varieties. Usually Frontana is very resistant, while Merit 3 and Premier are very susceptible to race 93; but the reverse is true on rare occasions. In the tests made during 1945, 1947, and 1948, Cadet invariably was highly susceptible to race 126, but a biotype of this race, used in 1946, failed to produce normal infection on seedlings of Cadet.

Mindful of the existence of biotypes among certain, or all, physiologic races and of their infrequent detection, a single seedling test cannot be considered an infallible indication of the varietal reaction to any specific leaf rust race. Consequently, more than one isolate of any given physiologic race should be used if the results obtained are expected to be reliable or significant.

Table 2. Seedling reaction to physiologic races of Puccinia rubigo-vera tritici of different varieties of wheat tested under greenhouse conditions.

Varieties tested		Reaction to specified races	
Name	C. I. No.	Resistant	Susceptible
Acme	5284	9, 10, 15, 31, 52, 77	1, 2, 3, 5, 7, 11, 12, 13, 14, 16, 17, 18, 20, 21, 28, 35, 49, 58, 90, 93, 126, 128
American Banner	6943		1, 2, 5, 9, 11, 12, 15, 21, 28, 31, 35, 52, 77, 91, 93, 126, 128
Aniversario	12578	3, 5, 9, 14, 16, 58, 90, 93, 126, 128	12
Apulia x Progreso	12587	1, 2, 3, 5, 9, 11, 12, 14, 15, 16, 21, 28, 31, 35, 43, 52, 58, 90, 93, 107, 126, 128	
Arnautka	1493	1, 2, 3, 5, 7, 9, 10, 11, 13, 15, 16, 17, 18, 20, 21, 28, 31, 35, 49, 52, 58, 77, 90, 93, 126, 128	12, 14
Australith	12808	21, 28	2, 3, 5, 7, 9, 11, 12, 14, 15, 16, 31, 35, 58, 90, 93, 126, 128
Baart-1121 x 1581 (N.No. 1919)	12877	1, 2, 7, 15	10, 17, 18, 31, 35, 49, 52, 77, 126, 128
Bahiense (N.S.No. [a] (III-46-16)	12591	28, 93	2, 3, 5, 7, 9, 11, 12, 13, 14, 15, 16, 31, 35, 58, 90, 126, 128
Baldrock	11538		1, 2, 5, 9, 11, 12, 15, 21, 28, 31, 35, 52, 77, 91, 93, 126, 128
Benvenuto-Inca	12588	1, 5, 11, 28	9, 12, 15, 21, 31, 35, 52, 93, 126, 128
Benvenuto Pampa	12809	1, 5, 9, 11, 28	12, 15, 21, 31, 35, 52, 93, 126, 128
Blue Jacket	12502		2, 3, 5, 9, 11, 12, 14, 15, 16, 58, 90, 93, 126, 128
Brevit	3778	1, 2, 3, 5, 7, 9, 13, 15, 16, 17, 18, 19, 21, 28, 31, 33, 34, 35, 37, 40, 44, 46, 52, 60, 90, 93, 126, 128	4, 6, 8, 10, 11, 12, 14, 20, 26, 29, 32, 42, 43, 49, 50, 56, 58, 61, 64, 77, 80, 91, 105, 107

[a]Minnesota Nursery stock number.

Table 2, (Continued)

Varieties tested		Reaction to specified races	
Name	C.I. No.	Resistant	Susceptible
Buck-Quequen	12574		1, 5, 9, 11, 12, 15, 21, 28, 31, 35, 52, 93, 126, 128
Cadet	12053	1, 2, 3, 5, 7, 9, 11, 15, 20, 26, 28	10, 12, 13, 14, 16, 17, 18, 21, 29, 31, 33, 35, 49, 52, 58, 61, 77, 90, 93, 107, 126, 128
Capelli	12452	1, 2, 3, 5, 7, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 21, 28, 31, 35, 43, 49, 52, 58, 77, 90, 93, 107, 126, 128	
Carina	3756	1, 2, 3, 5, 6, 7, 8, 9, 11, 14, 15, 16, 17, 28, 31, 32, 33, 34, 35, 37, 44, 50, 52, 58, 60, 61, 64, 90, 93, 105, 126, 128	4, 10, 12, 13, 18, 19, 20, 21, 26, 29, 40, 42, 43, 46, 49, 56, 77, 80, 91, 107
Carleton	12064	1, 2, 3, 5, 7, 9, 10, 11, 13, 14, 15, 16, 17, 18, 20, 21, 28, 31, 33, 35, 49, 52, 77, 90, 93, 126	12, 58, 61, 107, 128
Centenario	12021	1, 2, 3, 5, 9, 11, 12, 14, 15, 16, 20, 26, 28, 31, 33, 49, 58, 61, 107, 126	7, 21, 35, 52, 90, 93, 128
Ceres	6900		1, 2, 5, 9, 15, 20, 31, 33, 49
Ceres x Hope-Florence	11640		1, 2, 5, 9, 15, 31, 33
Ceres x Hope-Florence	11714		1, 2, 5, 9, 15, 31, 33
Cincana (N.S.No. III-47-19)	12810	2, 5, 11, 13, 28, 90, 93	3, 7, 9, 12, 14, 15, 16, 31, 35, 58, 126, 128
Democrat	3384	1, 4, 8, 9, 10, 11, 13, 14, 16, 17, 18, 19, 20, 26, 29, 31, 37, 43, 49, 50, 56, 64, 91, 93, 107, 128	2, 3, 5, 6, 7, 12, 15, 21, 28 32, 33, 34, 35, 40, 42, 44, 46, 52, 58, 60, 61, 77, 80, 90, 105, 126
Einkorn	2433	1, 2, 5, 6, 8, 9, 12, 13, 14, 15, 16, 20, 21, 31, 33, 35, 43, 44, 49, 50, 52, 60, 64, 90, 93, 126, 128	11, 28,
Esteana (N.S.No. III-46-12)	12811	9, 11, 13, 14, 16, 28, 31, 93, 128	2, 3, 5, 7, 12, 15, 35, 58, 90, 126
Eureka (N.S.No. III-46-15) (from Argentina)	12812	93	2, 3, 5, 7, 9, 11, 12, 13, 14 15, 16, 28, 31, 35, 58, 90, 126, 128
Exchange	12635	1, 2, 3, 4, 5, 7, 9, 11, 12,	

Table 2. (Continued)

Varieties tested		Reaction to specified races	
Name	C.I. No.	Resistant	Susceptible
Exchange (Continued)		14, 15, 16, 17, 19, 21, 28, 31, 33, 35, 37, 40, 43, 52, 58, 77, 90, 91, 93, 105, 107, 126, 128	
Frondoso	12078	1, 2, 5, 6, 11, 17, 31, 40, 50, 64, 91	9, 12, 15, 21, 28, 35, 49, 52, 77, 93, 126, 128
Frondoso x Chinese Progress (N.S.No. III-46-14)	12813	2, 5, 11, 14, 15, 93	3, 9, 12, 13, 16, 21, 28, 31, 35, 58, 90, 126, 128
(N.S.No. III-46-15)	12814	11, 12, 14, 21, 58, 93, 126, 128	2, 3, 5, 7, 9, 13, 15, 16, 28, 31, 35, 90
(N.S.No. III-46-16)	12815	11, 21, 93	2, 3, 5, 7, 9, 12, 13, 14, 15, 16, 28, 31, 35, 58, 90, 126, 128
Frontana	12470	1, 2, 3, 4, 5, 7, 9, 11, 12, 13, 14, 15, 16, 17, 19, 21, 28, 31, 33, 35, 37, 40, 43, 52, 58, 77, 90, 91, 93, 105, 126, 128	107
Fronteira	12019	93	1, 2, 3, 5, 7, 9, 11, 12, 14, 15, 16, 17, 21, 28, 31, 35, 52, 58, 90, 126, 128
Gabo	12795	1, 3, 4, 5, 7, 9, 15, 17, 19, 28, 31, 33, 35, 37, 40, 52, 58, 77, 91, 105, 126	
Gaza	12616	1, 2, 3, 5, 9, 13, 14, 15, 16, 21, 58, 90, 93, 126, 128	12, 28, 31, 35, 52
H-44	8177		1, 2, 5, 9, 15, 31, 33
Haynes Bluestem	2874	1, 5, 11, 15, 28, 52, 126, 128	9, 12, 21, 31, 35, 93
Henry	12265	1, 2, 3, 5, 7, 9, 10, 11, 12, 14, 15, 16, 20, 21, 26, 28, 33, 43, 49, 58, 61, 90, 91, 107, 126	17, 18, 31, 35, 52, 77, 93, 128
Hood	11456		2, 5, 6, 8, 9, 11, 13, 14, 15, 16, 20, 28, 31, 43, 44, 50, 60, 64, 90, 93
Hope	8178	3, 11, 12, 13, 35, 44, 52, 58, 64, 93	1, 2, 5, 6, 7, 8, 9, 14, 15, 16, 20, 21, 28, 31, 33, 43, 50, 60, 90, 126, 128
Hussar	4843	1, 3, 4, 5, 7, 8, 9, 10, 11, 15, 16, 19, 32, 33, 35, 37, 43, 44, 46, 50, 58, 90, 93, 105, 126	2, 6, 12, 13, 14, 17, 18, 20, 21, 28, 29, 31, 34, 40, 42, 49, 52, 56, 60, 61, 64, 77, 80, 91, 107, 128

Table 2. (Continued)

Varieties tested		Reaction to specified races	
Name	C.I. No.	Resistant	Susceptible
(Ill. No. 1-Chinese) ^{2} x Timopheevi (Wis. 245)	12633	9, 11, 12, 14, 15, 16, 58, 126	2, 3, 5, 90, 93, 128
Jenkin	5177		1, 2, 5, 9, 15, 31, 33
Kenya 58	12471		2, 3, 5, 9, 11, 12, 14, 15, 16, 58, 90, 93, 126, 128
Kenya 117-A	12568		2, 3, 5, 9, 11, 12, 14, 15, 16, 58, 90, 93, 126, 128
Khapli	4013	1, 2, 11, 15, 16, 28, 52, 58, 90, 93, 126, 128	3, 5, 9, 12, 14, 20, 21, 31, 35, 49, 61
Klein 66	12091		1, 5, 9, 11, 12, 15, 21, 28 31, 35, 52, 93, 126, 128
Klein 157	12586	1, 9, 11, 28, 31, 128	5, 12, 15, 21, 35, 52, 93, 126
Klein-Amalia	12577	1, 2, 3, 5, 11, 12, 14, 15, 16, 58, 93	9, 13, 21, 28, 31, 35, 52, 90, 126, 128
Klein-Exito	12581	1, 11	5, 9, 12, 15, 21, 28, 31, 35, 52, 93, 126, 128
Klein-Otto Wulff	12583	1, 11, 28	5, 9, 12, 15, 21, 31, 35, 52, 93, 126, 128
Klein-Sinmarq	12584	1, 11, 28	5, 9, 12, 15, 21, 31, 35, 52, 93, 126, 128
Klein Titan	12615	1, 3, 4, 5, 7, 9, 15, 17, 19, 28, 31, 33, 35, 37, 40, 52, 58, 77, 91, 105, 126	
Kota	5878		1, 2, 3, 5, 7, 9, 11, 12, 13, 14, 15, 16, 20, 21, 28, 31, 35, 49, 52, 58, 90, 93, 126, 128
Kubanka	1440	2, 3, 5, 9, 10, 11, 13, 14, 18, 31, 33, 52, 126, 128	1, 7, 12, 15, 16, 17, 20, 21, 28, 35, 49, 58, 77, 90, 93
Lageadinho (N.S.No. III-47-18)	12816	13, 15, 28, 31, 35	2, 3, 5, 7, 9, 11, 12, 14, 16, 58, 90, 93, 126, 128
La Prevision 25	12596	1, 2, 3, 4, 5, 7, 9, 11, 12, 14, 15, 16, 17, 19, 21, 28, 31, 33, 35, 37, 40, 43, 52, 58, 77, 90, 91, 93, 105, 107, 126, 128	
Lee	12488	1, 2, 3, 4, 5, 6, 7, 9, 11, 13, 14, 15, 16, 17, 19, 21, 28, 31, 33, 35, 37, 40, 43, 49,	12

Table 2. (Continued)

Varieties tested		Reaction to specified races	
Name	C.I. No.	Resistant	Susceptible
Lee (Continued)		50, 52, 58, 64, 77, 90, 91, 93, 105, 107, 126, 128	
Little Club	4066		2, 5, 9, 12, 15, 20, 28, 33, 49, 52, 61, 107, 128
Loros	3779	1, 2, 5, 7, 15, 16, 17, 34, 52, 56, 60, 91, 93	3, 4, 6, 8, 9, 10, 11, 12, 13, 14, 18, 19, 20, 21, 28, 29, 31, 32, 33, 35, 37, 40, 42, 43, 44, 46, 49, 50, 58, 61, 64, 77, 80, 90, 105, 107, 126, 128
M. A. 38 (N.S.No. III-46-2)	12597	2, 5, 15	3, 9, 11, 12, 13, 14, 16, 21, 28, 31, 35, 58, 90, 93, 126, 128
Malakof	4898	1, 2, 3, 4, 11, 12, 14, 15, 16, 18, 32, 33, 34, 44, 46, 56, 58, 61, 90, 107	5, 6, 7, 8, 9, 10, 13, 17, 19, 20, 21, 28, 29, 31, 35, 37, 40, 42, 43, 49, 50, 52, 60, 64, 77, 80, 91, 93, 105, 126, 128
Maroqanith	12817	5, 7, 13, 21, 35	2, 3, 9, 11, 12, 14, 15, 16, 28, 31, 58, 90, 93, 126, 128
Marquis	3641		1, 2, 3, 5, 7, 9, 11, 12, 13, 14, 15, 16, 21, 28, 31, 33, 35, 52, 58, 90, 93, 126, 128
McMurachy-Exchange x Redman (R.L. 2325) (N.S.No. III-46-17)	12832	1, 2, 3, 5, 7, 9, 11, 12, 13, 14, 15, 16, 21, 28, 31, 35, 43, 52, 58, 90, 93, 107, 126, 128	
McMurachy-Exchange x Redman (R.L. 2327)	12833	1, 5, 9, 11, 12, 15, 16, 21, 28, 31, 35, 43, 52, 93, 107, 126, 128	
Mediterranean	3332	1, 9, 10, 11, 13, 14, 16, 17, 18, 19, 20, 26, 29, 31, 33, 37, 43, 46, 49, 50, 64, 91, 93, 107, 128	2, 3, 4, 5, 6, 7, 8, 12, 15, 21, 28, 32, 34, 35, 40, 42, 44, 52, 56, 58, 60, 61, 77, 80, 90, 105, 126
Merit 3	12036	1, 2, 5, 7, 9, 11, 15, 20, 26, 28, 58, 90	3, 10, 12, 13, 14, 16, 18, 21, 31, 35, 49, 52, 61, 77, 93, 107, 126, 128
Mida	12008	1, 2, 5, 6, 7, 9, 11, 12, 15, 16, 20, 26, 28, 40, 50, 58, 64, 90, 91, 126	3, 10, 13, 14, 17, 18, 21, 31, 33, 35, 49, 52, 61, 77, 93, 107, 128
Mindum	5296	1, 2, 5, 7, 9, 10, 11, 13, 15, 16, 17, 18, 20, 21, 26, 28,	3, 12, 14, 61, 90, 107, 128

Table 2. (Continued)

Varieties tested		Reaction to specified races	
Name	C. I. No.	Resistant	Susceptible
Mindum (Continued)		31, 33, 35, 49, 52, 58, 77, 93, 126	
Newthatch	12318	1, 2, 5, 9, 11, 15, 20, 26, 28, 43, 58, 91	3, 7, 10, 12, 13, 14, 16, 17, 18, 21, 31, 33, 35, 49, 52, 61, 77, 90, 93, 107, 126, 128
Nursith	12818	2, 9, 13, 21, 28, 90, 93, 126, 128	3, 5, 11, 12, 14, 15, 31, 35, 58
Pelon Plateado	12819	1, 9, 11, 15, 28	5, 12, 21, 31, 35, 52, 93, 126, 128
Pentad	3320	5, 9, 21, 52, 126	1, 2, 3, 7, 11, 12, 14, 15, 28, 31, 35, 58, 90, 93, 128
Petiso	12820	1, 5, 9, 11, 15, 28	12, 21, 31, 35, 52, 93, 126, 128
Pilot	11945	1, 2, 3, 5, 6, 7, 9, 11, 15, 20, 26, 28, 40, 50, 58, 64, 90, 91	10, 12, 14, 16, 17, 18, 21, 31, 33, 35, 49, 52, 61, 77, 93, 107, 126, 128
Pilot x 1514	12476	1, 2, 5, 6, 11, 17, 31, 40, 50, 91	15, 35, 49, 64, 77, 128
Premier	11940	1, 2, 3, 12, 16, 31, 58, 90	5, 9, 11, 14, 15, 21, 28, 35, 52, 93, 126, 128
Premier x Bobin 2-Gaza (N.S.No. II-39-2)	12821	1, 2, 3, 5, 6, 7, 9, 11, 12, 13, 14, 15, 16, 17, 21, 28, 31, 35, 40, 43, 49, 50, 52, 58, 64, 77, 90, 91, 93, 107, 126, 128	
Quanah	12145	11	5, 9, 15, 52, 126
Red Egyptian	12345	1, 2, 5, 9, 11, 15, 20, 26, 28, 90,	3, 12, 14, 16, 21, 31, 33, 35, 43, 49, 52, 58, 61, 77, 93, 107, 126, 128
Regent	12070	1, 2, 3, 5, 7, 9, 11, 15, 20, 26, 28	10, 12, 14, 16, 17, 18, 21, 31, 33, 35, 49, 52, 58, 61, 77, 90, 93, 107, 126, 128
Reliance	7370		1, 2, 3, 5, 9, 11, 12, 14, 15, 16, 20, 21, 28, 31, 35, 49, 52, 58, 90, 93, 126, 128
Reliance-Hope x Pilot	12366	1, 2, 7, 15	10, 17, 18, 31, 35, 49, 52, 64, 126, 128
Reliance x R.L. 729-R. L. 2088 (N.S. No. III-47-36)	12822	2, 16, 21, 93, 128	3, 5, 9, 11, 12, 13, 14, 15, 28, 31, 35, 58, 90, 126

Table 2. (Continued)

Varieties tested		Reaction to specified races	
Name	C.I. No.	Resistant	Susceptible
Renown	11947	1, 2, 3, 5, 7, 9, 11, 15, 20, 28, 58, 90	10, 12, 14, 16, 17, 18, 21, 31, 33, 35, 49, 52, 61, 77, 93, 107, 126, 128
Reward	8182		1, 2, 5, 9, 15, 20, 31, 33, 49
Rio Negro	12469	1, 3, 5, 15, 17, 19, 28, 31, 35, 40, 52, 91	4, 7, 9, 33, 77, 126
Rival	11708	1, 2, 3, 5, 9, 11, 15, 20, 28, 58, 90	10, 12, 14, 16, 17, 18, 21, 31, 33, 35, 49, 52, 61, 93, 107, 126, 128
Saunders	12567	3, 15, 21, 28, 31, 35, 58, 90	2, 5, 9, 11, 12, 14, 16, 93, 126, 128
Sinvalocho	12595	1, 9, 28	5, 11, 12, 15, 21, 31, 35, 52, 93, 126, 128
Spelmar	6236	1, 2, 5, 7, 9, 11, 13, 14, 15, 16, 17, 18, 20, 21, 28, 31, 35, 52, 58, 77, 90, 93, 126, 128	3, 12, 49
Spinkcota	12499	1, 11, 15, 17, 31	2, 5, 6, 35, 40, 49, 50, 64, 77, 91, 128
Stewart	12066	2, 5, 10, 13, 20, 26, 28, 31	1, 3, 7, 9, 11, 12, 14, 15, 16, 17, 18, 21, 33, 35, 49, 52, 58, 61, 77, 90, 93, 107, 126, 128
Surpresa	12474	1, 2, 3, 5, 6, 9, 11, 14, 15, 16, 31, 40, 50, 58, 64, 90, 91, 93	12, 21, 28, 35, 52, 77, 126, 128
Thatcher	10003	44	1, 2, 5, 6, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 20, 21, 26, 28, 31, 33, 35, 40, 43, 49, 50, 52, 60, 61, 64, 77, 90, 91, 93, 107, 126, 128
Thew	5002	1, 58, 91	2, 3, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 20, 21, 26, 28, 31, 33, 35, 37, 40, 43, 44, 49, 50, 52, 60, 61, 64, 77, 90, 93, 105, 107, 126, 128
Timopheevi	11802	1, 2, 5, 9, 11, 12, 14, 15, 16, 20, 21, 26, 28, 31, 33, 35, 43, 49, 52, 58, 61, 90, 93, 107, 126, 128	

Table 2. (Concluded)

Varieties tested		Reaction to specified races	
Name	C.I. No.	Resistant	Susceptible
Timstein	12347	1, 2, 3, 5, 9, 11, 14, 15, 16, 20, 21, 26, 28, 33, 35, 52, 58, 90, 93, 126, 128	12, 31, 49, 61, 107
Timstein x Newthatch	12634	1, 2, 3, 5, 7, 9, 11, 12, 13, 14, 15, 16, 21, 28, 31, 35, 43, 52, 58, 90, 93, 107, 126, 128	
Trintecinco x Litoral (N.S.No. III-46-3)	12823	7, 13, 31,	2, 3, 5, 9, 11, 12, 14, 15, 16, 21, 28, 33, 35, 58, 90, 93, 126, 128
Triumph	12132		2, 3, 5, 9, 11, 12, 14, 15, 16, 58, 90, 93, 126, 128
Uruguay 2762/562003	12824	5, 9, 11, 15, 28, 31	1, 12, 21, 35, 52, 93, 126, 128
Uruguay 2762/5637	12825	5, 9, 11, 15, 28, 31	1, 12, 21, 35, 52, 93, 126, 128
Vernal	3686	1, 11, 28, 52	2, 3, 5, 9, 12, 14, 15, 16, 20, 21, 31, 35, 49, 58, 90, 93, 126, 128
Vernum	12255	2, 3, 5, 9, 11, 15, 90, 93 126, 128	12, 14, 58
Wabash x American Banner--outcross	12878	2, 3, 5, 9, 11, 12, 14, 15, 16, 43, 58, 90, 93, 107, 126, 128	
Warden x Leap	12660	2, 3, 5, 9, 11, 12, 14, 15, 16, 58, 90, 93, 107, 126, 128	43
Warden x Purkof	12879	2, 3, 5, 9, 11, 12, 14, 15, 16, 43, 58, 90, 93, 107, 126, 128	
Webster	3780	1, 2, 3, 4, 5, 6, 11, 12, 14, 15, 16, 17, 26, 28, 32, 33, 34, 37, 40, 43, 44, 46, 49, 50, 52, 56, 58, 61, 64, 93, 105, 126, 128	7, 8, 9, 10, 13, 18, 19, 20, 21, 29, 31, 35, 42, 60, 77, 80, 90, 91, 107
Yorkwin	11855		1, 2, 5, 9, 11, 12, 15, 21, 28, 31, 35, 52, 77, 91, 93, 126, 128

Table 3. Comparative reaction to leaf rust of wheat and durum varieties grown in field plots at University Farm, St. Paul, Minnesota.

Varieties tested		Percentage of infection in any given year									
Name	C.I. No.	1947	1946	1945	1944	1943	1942	1941	1940	1939	1938
Acme	5284	--	--	5	0	0	2	2	T	0	0
Arnautka	1493	T	T	5	15	0	T	T	1	0	--
Bahiense (N.S. No. III-46-16)	12591	10	--	--	--	--	--	--	--	--	--
Benvenuto-Inca	12588	10	--	--	10	10	--	--	--	--	--
Brevit	3778	--	--	90	--	--	--	--	--	--	--
Buck-Quequen	12574	T	--	--	10	5	--	--	--	--	--
Cadet	12053	40	50	65	60	T	5	1	--	--	--
Carina	3756	--	15	70	--	--	--	--	--	--	35
Carleton	12064	T	T	5	10	T	T	T	--	--	--
Cincana (N.S. No. III-47-19)	12810	10	10	--	--	--	--	--	--	--	--
Ceres	6900	80	50	70	100	50	60	40	60	75	80
Esteana (N.S. No. III-46-12)	12811	T	T	--	--	--	--	--	--	--	--
Eureka (N.S. No. III-46-15)	12812	60	--	--	--	60	--	--	--	--	--
Frondoso	12078	--	--	--	--	T	--	--	--	--	--
Frondoso x Chinese Progress (N.S. No. III-46-14)	12813	T	5	--	--	--	--	--	--	--	--
(N.S. No. III-46-15)	12814	10	5	--	--	--	--	--	--	--	--
(N.S. No. III-46-16)	12815	T	5	--	--	--	--	--	--	--	--
Frontana	12470	T	0	--	--	--	--	--	--	--	--
Fronteira	12019	--	0	20	--	T	--	--	--	--	--
H-44	8177	--	--	80	--	T	--	--	--	--	--
Haynes Bluestem	2874	60	50	80	80	35	42	50	25	80	80
Henry	12265	35	25	75	25	T	T	--	--	--	--
Hope	8178	50	50	80	50	T	T	T	T	T	T
Kenya 58	12471	--	80	50	57	--	--	--	--	--	--
Kenya 117A	12568	--	80	50	57	--	--	--	--	--	--
Klein 66	12091	--	--	--	10	T	--	--	--	--	--
Klein 157	12586	15	--	--	10	35	--	--	--	--	--
Klein-Amalia	12577	10	--	--	30	T	--	--	--	--	--
Klein-Exito	12581	10	--	--	70	10	--	--	--	--	--
Klein-Otto Wulff	12583	T	--	--	10	10	--	T	--	T	--
Klein-Sinmarq	12584	10	--	--	55	15	--	--	--	--	--
Klein Titan	12615	T	--	--	--	--	--	--	--	--	--
Kota	5878	75	75	70	95	50	95	95	100	65	75
Kubanka	1440	T	--	15	5	T	2	3	1	T	0
Lageadinho (N.S. No. III-47-18)	12816	T	--	--	--	--	--	--	--	--	--
La Prevision 25	12596	T	--	--	10	5	--	--	--	--	--
Lee	12488	T	10	35	--	--	--	--	--	--	--
Loros	3779	--	--	80	100	90	95	10	100	65	80
M. A.- 38 (N.S. No. III-46-2)	12597	10	T	--	--	--	--	--	--	--	--
Marquis	3641	85	60	60	90	60	50	60	80	80	80
McMurachy-Exchange x Redman (R.L. 2325)	12832	T	--	--	--	--	--	--	--	--	--
Merit 3	12036	--	--	--	--	85	3	1	10	5	--
Merit x Pilot	12315	30	25	60	--	--	--	--	--	--	--
Mida	12008	50	40	70	45	T	T	T	2	--	--
Mida x Cadet	12363	30	25	--	--	--	--	--	--	--	--
Mindum	5296	T	T	5	5	T	T	T	T	0	1

Table 3. (Concluded)

Varieties tested		Percentage of infection in any given year									
Name	C.I. No.	1947	1946	1945	1944	1943	1942	1941	1940	1939	1938
Newthatch	12318	80	70	80	65	T	T	T	T	--	--
Pentad	3320	5	T	5	10	0	3	3	T	T	1
Pilot	11945	40	40	65	60	1	T	T	2	30	10
Pilot x Mida	12303	28	25	--	--	--	--	--	--	--	--
Premier	11940	60	60	75	--	5	--	T	1	T	--
Premier x Bobin 2-Gaza (N.S. No. II-39-2)	12821	10	--	75	--	--	--	--	--	--	--
Preston	3081	65	65	45	85	35	45	95	95	45	65
Red Egyptian	12345	--	--	30	10	--	15	--	--	--	--
Regent	12070	70	65	70	75	T	T	T	25	T	--
Reliance	7370	65	50	65	80	20	30	70	45	45	30
Renown	11947	--	--	--	--	T	T	15	20	10	--
Reward	8182	--	--	--	--	--	--	--	--	--	100
Rio Negro	12469	T	T	--	--	--	--	--	--	--	--
Rival	11708	50	40	65	60	20	10	T	10	25	--
Rival x Thatcher	12273	73	--	--	--	--	--	--	--	--	--
Saunders	12567	80	--	--	--	--	--	--	--	--	--
Sinvalocho	12595	T	T	--	10	5	--	--	--	--	--
Spelmar	6236	T	T	5	5	T	T	T	1	0	1
Spinkcota	12499	--	70	80	--	--	--	--	--	--	--
Stewart	12066	T	T	5	20	T	T	1	--	--	--
Surpresa	12474	--	T	15	--	T	--	--	T	T	T
Thatcher	10003	80	70	90	90	60	75	100	80	80	80
Thew	5002	--	--	--	95	--	--	--	--	--	--
Timstein	12347	T	5	50	10	T	T	--	--	--	--
Webster	3780	5	20	30	10	15	20	60	5	20	15

COMPARATIVE VARIETAL REACTION TO LEAF RUST UNDER FIELD CONDITIONS

A total of 71 varieties and selections of hybrids of common and durum wheat were tested at University Farm, St. Paul, for their reaction to leaf rust in the maturing stage. About four-fifths of these varieties were grown in field plots under artificially induced leaf rust epidemics, superimposed on natural infection. The remainder constituted part of the uniform rust observation nurseries grown each year and were dependent on natural leaf rust epidemics for infection. Rust readings on 10 of the lines listed in Table 3 were available only for a single year, but were recorded in each of the 10 years for 13 other lines. Most of the common and durum varieties tested in the adult stage, under field conditions, were also tested in the seedling stage, under greenhouse conditions. The rust infections in the field plots were recorded in percentage terms based on the modified Cobb scale, generally used by cereal rust workers.

A perusal of the data presented in Table 3 will reveal that a number of varieties considered to be highly resistant to leaf rust until 1944 have shown very little if any resistance since that date. Notable among these varieties are: Cadet, Hope, Mida, Newthatch, Pilot, Regent, and, to a certain extent, Rival and Henry. Something like this was foreseen, when one of the authors of this report expressed his misgivings at the Seventh Hard Spring Conference held at St. Paul and Minneapolis on February 28 and 29 - March 1, 1944, in the following words: "It would seem obvious from the above records, meager as they may be, that there is not as yet, and perhaps never can be, any absolute assurance that a wheat variety highly resistant to leaf rust under a certain set of conditions in any given area over a limited period of time will not eventually prove to be very susceptible." That this would happen so soon afterwards could not be foreseen.

Although the tendency in recent years is rather general for wheat varieties to become more severely attacked by leaf rust, certain durum varieties, including Acme, Kubanka, Mindum, and Spelmar, seem to be as resistant now under field conditions as they were 35 years ago. On

the other hand, varieties such as Haynes Bluestem, Kota, and Preston, despite the fact that they have been practically out of cultivation on farms for from 15 to 25 years, have consistently been the subjects of leaf rust epidemics in disease gardens and uniform nurseries for 30 years or more. Several spring wheats, principally Frontana, Klein Titan, Lee, and McMurachy - Exchange x Redman, which have proved to be highly resistant under greenhouse conditions to a great number of physiologic races in the seedling stage and in many field tests also in the maturing stage, are now being extensively used as parental material in the breeding programs pursued at Minnesota and elsewhere.

UNIVERSITY OF MINNESOTA AND U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, DIVISION OF CEREAL CROPS AND DISEASES

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 200

PLANT PATHOLOGICAL INVESTIGATION
IN THE UNITED STATES
III

Supplement 200 March 30, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

PLANT PATHOLOGICAL INVESTIGATION IN THE UNITED STATES III

Plant Disease Reporter
Supplement 200 March 30, 1951

CONTENTS

A RÉSUMÉ OF THE ACTIVITIES OF THE MYCOLOGICAL COLLECTIONS OF THE UNITED STATES DEPARTMENT OF AGRICULTURE, WITH A PHYTOPATHOLOGICAL SLANT, 1885-1950

John A. Stevenson

Effective work on plant diseases in the United States Department of Agriculture began in 1885 when F. Lampson-Scribner joined the Division of Botany to head up the newly established Section of Mycology, of which he was in the beginning the entire technical staff. In those far-off days mycology or "applied mycology" and plant pathology were for practical purposes synonymous, early work being largely confined to plant diseases due to parasitic fungi or assumed to be.

It should perhaps be noted in passing that Dr. Thomas Taylor, microscopist of the Department (1871-1896), gave some attention to plant diseases as part of his varied and often bizarre activities, even before the time of Scribner. For the purposes of this account, however, Taylor's studies can be ignored, as practically all other writers on the subject have consistently done, on the grounds that they were of no importance in themselves and without any value as a foundation on which to commence other work.

Obviously Scribner on taking up his duties in the new work took over from the general herbarium, composed largely of flowering plants and which had been building up since 1869, all available fungus specimens to be used as one of his "working tools", and this early collection became at once a repository for fungus materials studied in the Section of Mycology as attested by specimens still to be found in the Bureau of Plant Industry Mycological herbarium. The first published record to this effect appears in Scribner's report for 1887 where as Chief of the Section he notes an herbarium consisting of 9300 labelled fungus specimens mounted on 5572 sheets. He adds that "permanent mounts of microscopical preparations to the number of about 500 were made during the year." Furthermore, a collection of European fungi of the vine was presented by Professor P. Viala, a noted French plant pathologist who spent some time here collaborating with Scribner on grape diseases, and a series of specimens was deposited by W. W. Calkins and S. M. Tracy representing Florida and western collections.

The pathological collections (plant disease material) continued to be maintained and built up as an integral and important part of the work in plant pathology for reference and study purposes, as is evidenced by continued comments in the annual reports of the Department. In 1888, B. T. Galloway, Scribner's successor, writes that, "During the year many new and valuable additions have been made to the herbarium and the number of microscopic mounts of fungi also materially increased."

As recorded in his report for 1891 the fungus herbarium had reached a total of 16,397 specimens mounted on 7865 sheets, representing 779 genera and 6424 species, including the more important exsiccati. By this time, as he notes, "general care of the herbarium requires the work of one assistant, while indexing, filing, and general supervision of the literature consumes a considerable part of the time of another." In this report too, mention is first made of the herbarium catalogue of hosts and fungi, an unique feature then and now of the Bureau mycological collections.

In Galloway's succeeding annual reports, mention of the herbarium is brief and of a general nature, but it was evident that activities outlined in 1891 were continued with direct interest at all times on the part of the Chief of the Division and his staff in maintaining and building up the collections. By 1895, a crisis was reached, which Galloway, writing in later years (Phytopath. 18: 877. 1928) described as follows "The glamour of field service in phytopathology was irresistible so that our collections and herbaria were beginning to languish and our mycological technique becoming rusty. To meet the situation we tried various expedients and made numerous experiments. It was the conviction of my colleagues that our only hope was to find a man, rich in experience, and so wedded to mycology that nothing could swerve him from the beaten path. The experiment was made, but the man failed us".

In this extremity, Mrs. Flora W. Patterson was placed in charge of the fungus herbarium in 1896 with the title of Assistant Pathologist, later changed to Mycologist with the formation of the Bureau of Plant Industry in 1901, and for more than 28 years she devoted herself unceasingly to the building up of the collections. During Mrs. Patterson's regime the unit, operating as a part of the Division of Vegetable Physiology and Pathology and after 1901 as an office of the Bureau of Plant Industry, was designated as Pathological Collections, a designation later (1926) changed to Mycological Collections under which name the unit is at present working.

Previous to Mrs. Patterson's time the herbarium and supporting catalogues or indices were

an integral part of the Division of Vegetable Physiology and Pathology along lines already outlined. With Mrs. Patterson's coming or sometime thereafter the fungus herbarium took on a separate status and its history as such begins.

Mrs. Patterson was charged with the duty, as have been her successors, of maintaining and building up the pathological (or mycological) collections and supporting files of the Division of Vegetable Physiology and Pathology and in turn of the Bureau of Plant Industry to serve as a reference collection and source of information on the fungi in general and more particularly on those concerned as causative agents of plant diseases. The herbarium has been a place of deposit for fungus materials studied and reported on by Bureau workers in other Divisions as well as for similar specimens received from many other outside sources. In addition to the obvious duties of curating the herbarium, building up the supporting catalogues and other collateral files, and making an endless series of routine fungus identifications, the mycologist in charge and other staff members have through the years carried on mycological research (usually with a phytopathological bearing) in so far as the above necessary duties have permitted.

Mrs. Patterson and her associate of many years, Miss Vera K. Charles, carried on inspection of all plant materials imported by the Department of Agriculture at Washington for a number of years and particularly those handled by the Office of Plant Introduction of the Bureau. They studied and named the fungi intercepted on such material as a basis for possible quarantine action. These duties were taken over by the Federal Horticultural Board of the Department soon after the passage of the original plant quarantine act of 1912.

For more than half a century the Department of Agriculture has played an important role in the development of techniques for mushroom culture, first in the Division of Vegetable Physiology and Pathology (43) and then in the office or Section of Mycological Collections. Margaret Ferguson's paper (44), published as a joint contribution with the Botany Department of Cornell University, was the first to describe clearly and in detail methods of germinating the spores of cultivated mushrooms and of producing pure culture spawn. Ferguson's work was done under the leadership of B. M. Duggar who, while a collaborator of the Section, was the first in the United States to study the nutritional and other cultural requirements of the cultivated mushroom as well as methods of spawn making (42). He prepared a Farmer's Bulletin (41) on mushroom culture and acted as a consultant on this subject until about 1925. Meanwhile Patterson and her associates maintained an active interest in the problems of mushroom culture as evidenced by the papers of Veihmeyer (123), Charles (17), Charles and Lambert (27), and Charles and Popenoe (28).

Serious and uncontrolled outbreaks of the Mycogone disease in the houses of commercial growers in Chester County, Pennsylvania, led to the establishment in 1928 of a project devoted entirely to the problems of the mushroom industry. This project was headed by E. B. Lambert until its temporary abandonment in 1943. In spite of limited funds the mushroom project was vigorously and fruitfully pursued during this period. A mushroom house was established at Arlington Farm, Virginia, and experimental investigations were carried out on all phases of mushroom culture. Published accounts of this work include papers on the control of Mycogone disease (69), the truffle disease (40), the rose comb disease (70), the plaster molds (27), monosporous cultures of the mushroom (68), plot technique for yield tests (72), casing soil practice (78), and the principles underlying composting practices (74, 75, 77). A critical survey of previous technical contributions in the field was published (73) in 1938, and two comprehensive accounts (71, 76) of mushroom growing with recommendations for improved cultural procedures were issued as Department publications.

To an even greater extent Patterson and Charles were concerned with the mushrooms of the fields and forests. They were continually called on for identification of poisonous and edible species and followed closely mushroom poisoning cases occurring in the local area. Their studies in this field were issued (89, 91) as Department publications which had a wide distribution.

Mrs. Patterson early in her scientific career became interested in the Exoascaceae, an important group of plant parasitic fungi. Her monographic study (85) of the group was published about the time of her coming to the Department. Unfortunately a press of other duties prevented her from pursuing her studies further with this group. She contributed a section on diseases to a Farmer's Bulletin on rose growing (87). The further work of herself and Miss Charles on diseases of ornamentals have been reviewed elsewhere in this series (Plant Dis. Reporter Supp. 195: 438. 1950). Among her other contributions may be mentioned a paper (86) describing 17 new species of fungi parasitic on plants, a note on an edible smut, and again in cooperation with Miss Charles a bulletin (88) on pineapple black rot and a downy mildew of an imported sedge, and finally a paper (90) on the occurrence of bamboo smut in America.

After Mrs. Patterson's retirement Miss Charles continued her work with the wild mushrooms (18) and was in addition for many years a specialist in the identification of entomogenous fungi. She published a number of papers on the subject (21, 22, 25), an interest which culminated in her check list (24) of these fungi for North America. Among her other mycological interests were fungi attacking animals (19, 23) and various new or rare fungi which came to her attention (20, 26).

At the time of Mrs. Patterson's retirement in 1923 the office of Pathological Collections was combined with the Plant Disease Survey, the new unit becoming the Office of Plant Disease Survey and Pathological Collections, later changed to the present Division of Mycology and Disease Survey. Dr. C. L. Shear, upon becoming head of the new office in 1923, continued his mycological and phytopathological studies, the latter nearly always with a distinctly mycological flavor, not only until his own retirement in 1935 but for many years thereafter. His papers were many and varied, including a revision of his bulletin on cranberry diseases (109), general discussions such as his Mycology, Scientific and Otherwise (98), Condition and Needs of Systematic Mycology (103), Mycology in Relation to Human Pathology (93), and a series on mycological nomenclature (94, 99). He was particularly interested in the taxonomy and life histories of the Ascomycetes and their imperfect stages, and of his papers in this field, many with co-workers, there may be cited those dealing with Tryblidiella, Penicillium glaucum, Botryosphaeria, Godronia, Dothiora, Physalospora, and Pilacre (96, 97, 102, 104, 105, 106, 108, 110, 111).

His miscellaneous studies in the field were gathered into a series of papers (100) under the general title of Mycological Notes, and he had made a beginning in publishing the results of a life-long study of the Xylariaceae by papers (101) on the genus Hypoxylon. He took part in editing the English edition of the classic work of the Tulasnes, Selecta Fungorum Carpologia, in three large folio volumes, and collaborated with F. E. Clements in the preparation and publication of the Genera of the Fungi (31), the standard book in the field. Similar papers (92, 95, 107) dealing with the Texas root-rot fungus (Ozonium (Phymatotrichum) omnivorum), Sphaceloma ampelina, and the Monilia sitophila group are worthy of comment. The latter paper with B. O. Dodge touched off the genetical work with these forms, which has now mushroomed into hundreds of titles in the literature on the genetics of the fungi.

Dr. James R. Weir was in charge of the mycological herbarium for the period 1923-1927, but during much of this time was concerned with field studies of rubber and sugar cane diseases, activities outside the scope of this paper.

Studies on the genus Balansia, an important group of grass parasites, have been carried on by W. W. Diehl, with a series of papers culminating in a recent monograph (38). Other papers by Dr. Diehl of a plant pathological nature have included studies of the false smut of maize (Ustilaginoidea) (46), taxonomy of Peziza quernea, (39), the Myriogenospora disease of grasses (35), and a new Ophiobolus (84a) on eelgrass, the latter a contribution to the mysterious eelgrass disease problem. In the general field of mycology he has published on mycogeography (36), Fungi of the Wilkes Expedition (33), the genus Astrocystis (34), and has collaborated with the Cotton Division in studying fungi concerned in deterioration of fabrics (37, 125).

The genus Elsinoë and its imperfect stage Sphaceloma have had detailed attention from Dr. A. E. Jenkins over a period of many years. These fungi constitute a group of plant parasites of considerable importance which were obscure and little known until the work of Dr. Jenkins and her co-worker A. A. Bitancourt, now Director of the Biological Institute, São Paulo, Brazil. Their studies, both individual and joint, have brought to light many new species and resulted in a long series of papers among which there may be cited here those dealing with scab and similar diseases of citrus (1, 51, 55), avocado (54), Canavalia (48), Cinnamomum and Sesbania (82), lima bean (49), apple and pear (52), rose (53), violet (84), goldenrod (66), sweet potato (67), Capulin cherry (59), mango (5), tea (2, 64), and cinchona (48). Many other plants of less importance are attacked by members of this genus. A considerable series of additional papers under the joint authorship of these co-workers dealing with the Elsinoaceae may be cited (3, 4, 6, 60, 61, 62, 63, 65). These papers describe new species, present new records of geographical distribution, and make available new data on other phases of the problem. Jenkins and Bitancourt have issued, as a further contribution to the elucidation of this group of fungi, 500 selected specimens representing their work under the title Myriangiales Selecti Exsiccati, sets of which have been deposited in ten mycological institutions in Europe, Asia, North and South America.

Dr. Jenkins has been interested also in the genus Taphrina but to date has published only her studies on species attacking various species of Acer (56). Other studies have included currant cane blight, Sclerotinia disease of mulberry (112), leaf spot of Convallaria (57), and

brown canker (47, 50) and other rose diseases.

Alaskan fungi (8) and the Discomycetes or cup fungi have been the chief research interest of Miss E. K. Cash. Several of her papers (16, 32, 83) on the latter group have been in cooperation with members of the Division of Forest Pathology dealing with species parasitic on trees. Other papers (9, 10, 11, 13, 14) have been concerned with the Discomycetes of Colombia, California, Colorado, Florida, Georgia, and Hawaii. The same worker has also contributed papers on fungi parasitic on Smilax, Ribes, and Libocedrus (7, 12, 15). Particular reference must be made to a study (124), by Miss Waterman of the Division of Forest Pathology and Miss Cash, on a new Discomycete in the genus Septotinia, found in its conidial state causing a very severe leaf blight of hybrid poplars.

Mr. Paul Lentz has been studying the morphology of wood-rotting fungi in the genus Stereum and has published papers (80, 81) on the genus Marssonina on oak and chestnut and on the fungus collections of the late George W. Carver of Tuskegee Institute.

The writer has been involved in a variety of activities since joining the mycological work in 1927. Some of these have resulted in publications more or less pertinent to the present subject. Many of these have been in cooperation with workers in other Divisions of the Bureau, including studies on tobacco blue mold (29, 30), tomato fruit rot (120), cowpea leaf spot (79), and sugarcane fungi (121). A long-continued interest in the plant diseases of other countries resulted in a Department publication (113) on the subject, which is now sadly in need of revision. Annotated lists of the fungi and primarily the plant parasitic fungi of several countries have been brought together in publications (45, 118, 122) issued with collaboration abroad. Contributions to the fungus flora of Nevada (117), and the Shenandoah National Park (114) may also be recorded. With Miss Cash an extensive work (119) dealing with the new fungus names proposed by C. G. Lloyd, whose fungus herbarium forms part of the collections, was published through the Lloyd Library. Studies of parasitic fungi which appeared to be undescribed have been issued in a series under the general title of Fungi Novi Denominati (115). As an aid to stablilization of author's abbreviations used in connection with fungus names a preliminary list of recommended abbreviations was issued (116).

The mycological and pathological collections of the Bureau now consist of a general fungus herbarium with an added unit of virus and other non-mycological specimens, a series of special collections, supporting indexes, a microscopic slide collection, photograph and literature collections, historical reference materials and other pertinent mycological items. The several units total more than 500,000 specimens, thus ranking the collections as second in size in the United States, the Farlow Cryptogamic Herbarium of Harvard University taking first place and the New York Botanical Garden third.

In the general herbarium there has been deposited through the years much of the material worked over by the plant pathologists and mycologists of the Bureau and its predecessor, the Division of Vegetable Physiology and Pathology. Among those whose fungus collections, to a greater or less extent, will be found here are F. Lampson-Scribner, B. T. Galloway, H. J. Webber, M. B. Waite, Erwin F. Smith, W. T. Swingle, David Fairchild, P. H. Dorsett, M. A. Carleton, W. A. Orton, C. R. Ball, F. W. Patterson, C. L. Shear, Geo. G. Hedgcock, Vera K. Charles, N. E. Stevens, J. R. Weir, W. A. Archer, and A. G. Johnson, as well as many other past and present workers in this and the crop divisions of the Bureau.

Many important series of specimens have been received from outside sources by exchange, purchase, or donations, among which may be noted those of J. P. Anderson, Alaska; E. Bethel, Colorado and other western States; G. Bresadola, worldwide; W. W. Calkins, Florida; G. W. Carver, Alabama; F. D. Heald and F. A. Wolf, Texas; H. W. Ravenel, Texas (1869, probably formed the beginning of the herbarium); A. A. Heller, California and Puerto Rico; E. W. D. Holway, Iowa and South America; W. A. Kellerman, West Virginia and Guatemala; A. B. Langlois, Louisiana; J. Lind, Denmark; E. D. Merrill and O. A. Reinking, Philippine Islands; L. W. Nuttall, West Virginia; H. E. Parks, California; E. A. Rau, Pennsylvania; J. Rick, Brazil; A. B. Seymour, Illinois and Massachusetts; R. Sprague, Oregon and North Dakota; P. C. Standley, Central America; F. L. Stevens, Puerto Rico, British Guiana, Ecuador, Panama, Hawaii and the Philippine Islands; John A. Stevenson, Puerto Rico; Thomas Taylor, United States; B. C. Tharp, Texas; S. M. Tracy and F. S. Earle, Mississippi; replicates of the types and other collections of L. Fuckel from the Herbier Boissier, Geneva, Switzerland; fungi of the Wilkes expedition; fungi of the North Pacific Exploring Expedition.

For some years all published exsiccati received, both old and new, have been kept in the original volumes as issued or made up into bound volumes if received as unmounted specimens. This series now totals over 70,000 specimens and has been indexed for ease of reference.

The Forest Pathology herbarium is curated separately in cooperation with the Division of Forest Pathology and consists of some 29,000 specimens resulting from the Bureau's work in that

field. This collection is particularly rich in wood-rotting fungi and in the rust fungi of forest trees.

The fungus collections of the Department of Botany of the Smithsonian Institution, consisting of three units, are at Beltsville with the Bureau collections under a cooperative agreement. The most important of these is the C. G. Lloyd Mycological Collection of nearly 60,000 specimens representing the life work of the late C. G. Lloyd of Cincinnati. It includes an excellent representation of the larger fungi of the world, being particularly noteworthy for its Polyporaceae, Xylariaceae, and Gasteromycetes. In addition to the actual specimens this herbarium includes Mr. Lloyd's negatives (about 10,000), his note-books, mycological correspondence, microscopic mounts and related materials. The other Smithsonian units consist of a general collection and the W. H. Long collection of rusts and puffballs, the two totalling about 20,000 specimens.

Other portions of the Bureau collections maintained as separate units include (1), the George L. Zundel collection of smut fungi, (2) insect-attacking fungi (entomogenous), (3) the C. C. Plitt lichen herbarium, (4) the Chinese herbarium, consisting of that portion of the Chinese National fungus collections originally at Nanking, not destroyed by the Japanese, (5) herbarium of sugarcane fungi, (6) herbarium of rubber (Hevea) fungi, (7) the E. E. Morse collection of Pacific Coast fungi, (8) a collection of fungi and pathological specimens preserved in liquid, including a classical series of specimens of bacterial plant diseases prepared by Erwin F. Smith and his associates. Particular mention must be made of the Michener herbarium found many years ago in a Pennsylvania attic by C. L. Shear and N. E. Stevens. It consists in large part of portions of the original Schweinitz collections from Carolina and Pennsylvania which Ezra Michener was permitted to take as recompense for his labors in organizing the Schweinitz herbarium at the Philadelphia Academy of Sciences. Many specimens from the M. A. Curtis herbarium as well as Michener's own fungus collections are included.

For many years an alphabetical fungus catalogue has been maintained to facilitate reference to the general herbarium, the various exsiccati series and the several special collections. This is supplemented by a host catalogue. The Lloyd herbarium is catalogued separately. A third catalogue, which has been in progress for nearly 30 years and has reached substantial proportions, cites the place, date, and author of new fungus genera and species and includes new combinations and other nomenclatorial changes. A geographical index to fungi in the general herbarium from all South and Central American countries, Mexico, and the District of Columbia area is under way. Since 1927, the mycological and plant pathological literature of the world has been catalogued in so far as time has permitted. Although obviously far short of complete coverage this catalogue does afford an excellent cross section of published information on the fungi in this field.

The fungus collections, catalogues, reference materials, and all other mycological facilities of the section are readily available to all who may have need to use them.

SELECTED BIBLIOGRAPHY

1. Bitancourt, A. A., and A. E. Jenkins. Sweet orange fruit scab caused by Elsinoë australis. Jour. Agric. Res. 54: 1-18, illus. 1937.
2. _____, and _____. Elsinoë theae causing scab of tea. Arq. Inst. Biol. 10: 193-198, illus. 1939.
3. _____, and _____. Thirteen new species of Elsinoë from Brazil. Arq. Inst. Biol. 12: 1-20, illus. 1941.
4. _____, and _____. New discoveries of Myriangiales in the Americas. Proc. 8th Amer. Sci. Congress, pp. 149-172, illus. 1942.
5. _____, and _____. Mango scab. Arq. Inst. Biol. 17: 205-228, illus. 1946.
6. _____, and _____. Ten new species of Elsinoaceae discovered in Brazil. Arq. Inst. Biol. São Paulo 19: 93-110, illus. 1949.
7. Cash, E. K. Godronia urceolus and other Cenangiaceae on Ribes. Mycologia 26: 266-272, illus. 1934.
8. _____. Alaskan fungi. Plant Dis. Reptr. 18: 74-88; 20: 121-123. 1934, 1936.
9. _____. Some Ascomycetes new to California. Mycologia 28: 247-252, illus. 1936.
10. _____. Some new Colorado Discomycetes. Mycologia 28: 297-306, illus. 1936.
11. _____. New records of Hawaiian Discomycetes. Mycologia 30: 97-107, illus. 1938.

12. Cash, E. K. Two species of Hysteriales on Smilax. Mycologia 31: 289-296, illus. 1939.
13. _____. Some Georgia Discomycetes. Jour. Wash. Acad. Sci. 29: 47-51, illus. 30: 299-305. 1939, 1940.
14. _____. Some new or rare Florida Discomycetes and Hysteriales. Mycologia 35: 595-603, illus. 1943.
15. _____. Some new species of fungi on Libocedrus. Mycologia 37: 311-317, illus. 1945.
16. _____, and R. W. Davidson. Some new species of Ascomycetes on coniferous hosts. Mycologia 32: 728-735, illus. 1940.
17. Charles, V. K. Mushroom culture for amateurs. U. S. Dept. Agric. Farmers' Bull. 1587: 1-16, illus. 1929, 1933.
18. _____. Some common mushrooms and how to know them. U. S. Dept. Agric. Circ. 143: 1-59, illus. 1931.
19. _____. Microsporum of cats causing ringworm in man. Jour. Wash. Acad. Sci. 24: 222-227. 1934.
20. _____. A little known pecan fungus. Mycologia 27: 74-82, illus. 1935.
21. _____. A fungus on lace bugs. Mycologia 29: 216-221, illus. 1937.
22. _____. A new entomogenous fungus on the corn earworm. Phytopath. 28: 893-897, illus. 1938.
23. _____. A ringworm disease of muskrats transferable to man. Jour. Wash. Acad. Sci. 30: 338-344. 1940.
24. _____. A preliminary check list of the entomogenous fungi of North America. Insect Pest Survey Bull. 21: 707-785. 1941.
25. _____. A fungous disease of codling moth larvae. Mycologia 33: 344-349, illus 1941.
26. _____. A Laschia on cabbage palmetto. Mycologia 34: 235-240, illus. 1942.
27. _____, and E. B. Lambert. Plaster molds occurring in beds of the cultivated mushroom. Jour. Agric. Res. 46: 1089-1098, illus. 1933.
28. _____, and C. H. Popenoe. Some mushroom diseases and their carriers. U. S. Dept. Agric. Circ. 27: 1-9, illus. 1928.
29. Clayton, E. E., and John A. Stevenson. Nomenclature of the tobacco downy mildew fungus. Phytopath. 25: 516-521, illus. 1935.
30. _____, and _____. Peronospora tabacina, the organism causing blue mold disease of tobacco. Phytopath. 33: 101-113. 1943.
31. Clements, F. E., and C. L. Shear. The genera of fungi. New York. 1931.
32. Davidson, Ross W., and E. K. Cash. Species of Sclerotinia from Grand Mesa National Forest. Mycologia 25: 266-273, illus. 1933.
33. Diehl, W. W. The fungi of the Wilkes Expedition. Mycologia 13: 38-41. 1921.
34. _____. The genus Astrocystis. Mycologia 17: 185-190, illus. 1925.
35. _____. The Myriogenospora disease of grasses. Phytopath. 24: 677-681, illus. 1934.
36. _____. A basis for mycogeography. Jour. Wash. Acad. Sci. 27: 244-254. 1937.
37. _____. Concerning the identity of Iterson's cellulolytic Mycogone. Mycologia 41: 277-279, illus. 1949.
38. _____. Balansia and the Balansiae, in America. U. S. Dept. Agric. Monograph 4: 1-82, illus. 1950.
39. _____, and E. K. Cash. The taxonomy of Peziza quernea. Mycologia 21: 243-245, illus. 1929.
40. _____, and E. B. Lambert. A new truffle in beds of cultivated mushrooms. Mycologia 22: 223-226, illus. 1930.
41. Duggar, B. M. The cultivation of mushrooms. U. S. Dept. Agric. Farmers' Bull. 204: 1-24, illus. 1904.
42. _____. The principles of mushroom growing and mushroom spawn making. Bur. Pl. Ind. Bull. 85: 1-60, illus. 1905.
43. Falconer, Wm. How to grow mushrooms. U. S. Dept. Agric. Farmers' Bull. 53: 1-19, illus. 1897.
44. Ferguson, Margaret C. A preliminary study of the germination of the spores of Agaricus campestris and other Basidiomycetous fungi. Bur. Pl. Ind. Bull. 16: 1-40, illus. 1902.

45. Garcia Rada, G., and John A. Stevenson. La flora fungosa peruana. Lima, Peru. 112 pp. 1942.
46. Haskell, R. J., and W. W. Diehl. False smut of maize, Ustilaginoidea. Phytopath. 19:589-592, illus. 1929.
47. Jenkins, A. E. Brown canker of roses, caused by Diaporthe umbrina. Jour. Agric. Res. 15: 593-599, illus. 1918.
48. _____. Scab of Canavalia caused by Elsinoë canavaliae. Jour. Agric. Res. 42: 1-12, illus. 1931.
49. _____. Lima-bean scab caused by Elsinoë. Jour. Agric. Res. 42: 13-23, illus. 1936.
50. _____. Development of brown canker of roses. Jour. Agric. Res. 42: 293-299, illus. 1931.
51. _____. Development of the citrus-scab oragnism Sphaceloma fawcettii. Jour. Agric. Res. 42: 545-558, illus. 1931.
52. _____. Elsinoe on apple and pear. Jour. Agric. Res. 44: 689-700, illus. 1932.
53. _____. Rose anthracnose caused by Sphaceloma. Jour. Agric. Res. 45: 321-337, illus. 1932.
54. _____. Sphaceloma perseae the cause of avocado scab. Jour. Agric. Res. 49: 859-869, illus. 1934.
55. _____. Australian citrus scab caused by Sphaceloma fawcettii scabiosa. Phytopath. 26: 195-197, illus. 1936.
56. _____. A new host for Taphrina dearnessii and geographic distribution of Taphrina on North American maples. Mycologia 32: 408-414, illus. 1940.
57. _____. Ascochyta majalis identified on lily of the valley in the United States. Phytopatn. 32: 259-261, illus. 1942.
58. _____. Scab of cinchona in South America caused by Elsinoë. Jour. Wash. Acad. Sci. 35: 344-352, illus. 1945.
59. _____. A new species of Elsinoë on Capulin cherry. Jour. Wash. Acad. Sci. 37: 86-89, illus. 1947.
60. _____, and A. A. Bitancourt. Doenças das plantas causadas por fungos dos generos Elsinoë e Sphaceloma.
61. _____, and _____. Illustrations of South American Elsinoë and Sphaceloma diseases known up to January 1936. Arq. Inst. Biol. São Paulo 10: 31-60, illus. 1939.
62. _____, and _____. An Elsinoë causing an anthracnose of Virginia creeper. Phytopath. 32: 424-427, illus. 1942.
63. _____, and _____. A new species of Sphaceloma causing scab of plantain. Jour. Wash. Acad. Sci. 36: 225-227, illus. 1946.
64. _____, and _____. Two scab diseases of tea caused by "Elsinoë" and their distribution. Arq. Inst. Biol. São Paulo 17: 67-72, illus. 1946.
65. _____, and _____. Diagnosis of the Elsinoë of flowering dogwood. Jour. Wash. Acad. Sci. 38: 362-365, illus. 1948.
66. _____, and H. G. Ukkelberg. Scab of goldenrod caused by Elsinoë. Jour. Agric. Res. 51: 515-525, illus. 1935.
67. _____, and A. P. Viégas. Stem and foliage scab of sweet potato. Jour. Wash. Acad. Sci. 33: 244-249, illus. 1943.
68. Lambert, E. B. The production of normal sporophores in monosporous cultures of Agaricus campestris. Mycologia 21: 333-335, illus. 1929.
69. _____. Studies on the relation of temperature to the growth, parasitism, thermal death point, and control of Mycogone perniciosa. Phytopath. 20: 75-83, illus. 1930.
70. _____. Two new diseases of cultivated mushrooms. Phytopath. 20: 917-919, illus. 1930.
71. _____. Mushroom growing in the United States. U. S. Dept. Agric. Circ. 251: 1-31, illus. 1932.
72. _____. Size and arrangement of plots for yield tests with cultivated mushrooms. Jour. Agric. Res. 48: 971-980, illus. 1934.
73. _____. Principles and problems of mushroom culture. Bot. Review 4: 397-426, 1938.
74. _____. Indoor composting for mushroom culture. U. S. Dept. Agric. Circ. 609: 1-15, illus. 1941.
75. _____. Studies on the preparation of mushroom compost. Jour. Agric. Res. 62: 415-422. 1941.

76. Lambert, E. B. Mushroom growing in the United States. U. S. Dept. Agric. Farmers' Bull. 1875: 1-38, illus. 1941.
77. _____, and A. C. Davis. Distribution of oxygen and carbon dioxide in mushroom compost heaps as affecting microbial thermogenesis, acidity, and moisture therein. Jour. Agric. Res. 48: 587-601. 1934.
78. _____, and Harry Humfeld. Mushroom casing soil in relation to yield. U. S. Dept. Agric. Circ. 509: 1-11, illus. 1939.
79. Lefebvre, C. L., and J. A. Stevenson. The fungus causing zonate leafspot of cowpea. Mycologia 37: 37-45, illus. 1945.
80. Lentz, Paul L. The genus Marssonina on Quercus and Castanea. Mycologia 42: 259-264, illus. 1950.
81. _____, and John A. Stevenson. A resumé of the fungus collections of George W. Carver. Plant Dis. Reptr. 32: 195-215. 1948.
82. Limber, P. D., F. G. Pollack, and A. E. Jenkins. Elsinoë discovered on Sesbania and Cinnamomum in the United States. Mycologia 38: 463-472, illus. 1926.
83. Lohman, M. L., and E. K. Cash. Atropellis species from pine cankers in the United States. Jour. Wash. Acad. Sci. 30: 255-262, illus. 1946.
84. Massey, L. M., and A. E. Jenkins. Scab of violet caused by Sphaceloma. Cornell Univ. Agric. Exp. Sta. Mem. 176: 1-9, illus. 1935.
84a. Mounce, I. and W. W. Diehl. A new Ophiobolus on eelgrass. Canadian Jour. Res. 11: 242-246, illus. 1934.
85. Patterson, F. W. A study of North American parasitic Exoascaceae. Iowa Univ. Bull. Lab. Nat. Hist. 3: 89-135, illus. 1895..
86. _____. New species of fungi. Bull. Torr. Bot. Club 27: 282-286. 1900.
87. _____, in F. L. Mulford. Roses for the home. Farmers' Bull. 750: 32-35. 1916.
88. _____, and V. K. Charles. Some fungous diseases of economic importance. U. S. Dept. Agric. Bur. Plant Ind. Bull. 171: 1-41, illus. 1910.
89. _____, and _____. Mushrooms and other common fungi. U. S. Dept. Agric. Bull. 175: 1-64, illus. 1915.
90. _____, and _____. The occurrence of bamboo smut in America. Phytopath. 61: 351-356, illus. 1916.
91. _____, and _____. Some common edible and poisonous mushrooms. U. S. Dept. Agric. Farmers' Bull. 796: 1-24, illus. 1917.
92. Shear, C. L. The life history of the Texas root rot fungus. Ozonium omnivorum. Jour. Agric. Res. 30: 475-477, illus. 1925.
93. _____. Mycology in relation to human pathology. Amer. Nat. 61: 151-159. 1927.
94. _____. (1) Mycological nomenclature in relation to plant pathology. (2) Mycological nomenclature. Proc. 4th Int. Congress Plant Sci. 2: 1225-1226, 1657-1660. 1929.
95. _____. The life history of Sphaceloma ampelina. Phytopath. 19: 673-678, illus. 1929.
96. _____. Life histories of Tryblidiella species. Mycologia 25: 274-285, illus. 1933.
97. _____. Penicillium glaucum of Brefeld refound. Mycologia 26: 104-107, illus. 1934.
98. _____. Mycology, scientific and otherwise. Mycologia 26: 201-209. 1934.
99. _____. Uniformity and stability of mycological nomenclature . Mycologia 28: 337-346. 1936.
100. _____. Mycological notes I-IX. Mycologia 29: 355-363, 30: 580-593, 31: 322-336, 32: 105-111, 32: 541-549, 33: 318-332, 34: 263-273, 35: 469-476, 38: 664-673, 40: 748-759, illus. 1937-1948.
101. _____. Studies of types and authentic specimens of Hypoxylon I, II. Lloydia 8: 245-262, 10: 60-64. 1945, 1947.
102. _____, and H. F. Bain. Life history and pathological aspects of Godronia cassandrae on cranberry. Phytopath. 19: 1017-1024, illus. 1929.
103. _____, and F. E. Clements. The conditions and needs of systematic mycology. Science 431: 393-395. 1926.
104. _____, and Ross W. Davidson. The life histories of Botryosphaeria melanops and Massaria platani. Mycologia 28: 476-482, illus. 1936.

105. Shear, C. L., and Ross W. Davidson. A new species of Dothiora on aspen and willow. Mycologia 32: 105-111. 1940.
106. _____, and B. O. Dodge. The life history of Pilacre faginea (Fr.) Berk. & Br. Jour. Agric. Res. 30: 407-417, illus. 1925.
107. _____, and _____. Life histories and heterothallism of the red bread-mold fungi of the Monilia sitophila group. Jour. Agric. Res. 34: 1019-1042, illus. 1927.
108. _____, and N. E. Stevens. Botryosphaeria and Physalospora in the Hawaiian Islands. Mycologia 21: 313-320, illus. 1929.
109. _____, and _____, and H. F. Bain. Fungous diseases of the cultivated cranberry. U. S. Dept. Agric. Tech. Bull. 258: 1-57, illus. 1931.
110. _____, and _____, and Marguerite S. Wilcox. Botryosphaeria and Physalospora on currant and apple. Jour. Agric. Res. 28: 589-598, illus. 1924.
111. _____, _____, and Marguerite S. Wilcox. Botryosphaeria and Physalospora in the Eastern United States. Mycologia 17: 98-107, illus. 1925.
112. Siegler, E. A., and A. E. Jenkins. Sclerotinia carunculoides, the cause of a serious disease of the mulberry. Jour. Agric. Res. 23: 833-836, illus. 1923.
113. Stevenson, John A. Foreign plant diseases. U. S. Dept. Agric. Misc. Publ. 198 pp. 1926.
114. _____. A preliminary list of the fungi of the Shenandoah National Park, I, II. Claytonia 3: 21-28, 31-35. 1937.
115. _____. Fungi novi denominati - I, II. Mycologia 35: 629-637; 38: 524-533. 1943, 1946.
116. _____. A preliminary list of authors of plant parasites with recommended abbreviations. Plant Dis. Reptr. 28: 366-395. 1944.
117. _____, and W. A. Archer. A contribution to the fungus flora of Nevada. Plant Dis. Reptr. 24: 93-103, 26: 196-197. 1940, 1942.
118. _____, and Martin Cardenas. Lista preliminar de los hongos de Bolivia. Lilloa 21: 77-134. 1949.
119. _____, and E. K. Cash. The new fungus names proposed by C. G. Lloyd. Bull. Lloyd Library 35 (Myc. Ser. 8): 1-209. 1936.
120. _____, and L. P. McColloch. Myrothecium as a tomato fruit rot organism. Plant Dis. Reptr. 31: 147-149. 1947.
121. _____, and R. D. Rands. An annotated list of the fungi and bacteria associated with sugarcane and its products. Hawaiian Planter's Record 42: 247-313. 1938.
122. _____, and F. L. Wellman. A preliminary account of the plant diseases of El Salvador. Jour. Wash. Acad. Sci. 34: 259-268. 1944.
123. Veihmeyer, F. J. The Mycogone disease of mushrooms and its control. U. S. Dept. Agric. Bull. 127: 1-24, illus. 1914.
124. Waterman, Alma M., and E. K. Cash. Leaf blotch of poplar caused by a new species of Septotinia. Mycologia 42: 374-384, illus. 1950.
125. Zuck, R. K., and W. W. Diehl. On fungal damage to sun-exposed cotton duck. Amer. Jour. Bot. 33: 374-382, illus. 1946.

DIVISION OF MYCOLOGY AND DISEASE SURVEY

HISTORY OF PLANT DISEASE RESEARCH IN TEXAS

P. A. Young and G. M. Watkins

The practice of plant pathology in Texas has closely attended the varied interests of farm economy in the State. Much of the land is fairly flat and farming has developed in accordance with conditions of soil type and fertility and rainfall or irrigation water. Climatic variation from north to south and east to west strongly influences the selection of adapted crops. The northern part, known as the Panhandle, and the adjacent areas to the south are a part of the hard red winter wheat belt. In the extreme south a subtropical climate favors a large citrus industry and intensive production of vegetables in the winter. The eastern section rather closely resembles the States to the east in climate, terrain and soils, with the result that the crops grown are similar to those of most Southeastern States, except that tobacco has never established a prominent position in Texas farming. Amid the western grazing lands the development of several important irrigated valleys has extended crop production to the most westward portion of the State, the El Paso Valley along the Rio Grande. Throughout the central part the areas adapted to cultivation are devoted principally to cotton, sorghums, corn, and small grains, with several legumes in use for forage and soil-building. Along the Gulf Coast from Orange to Corpus Christi a belt of flat land about one hundred miles wide, composed of soils generally very high in fertility, is devoted chiefly to rice, cotton, sorghums, and winter vegetables. Departures from the general crop interests of the various sections of the State are noted near the large cities, especially Houston, Dallas, Fort Worth, San Antonio, and El Paso, each of which supports considerable nearby market gardening. In other areas, such as districts in the Panhandle and the adjacent south plains, a combination of abundant water for irrigation and suitable soil qualities is favorable for the production of potatoes and other truck crops.

Considering the entire State, the principal crops are cotton, wheat, flax, sorghums, oats, rice, barley, corn, peanuts, clovers, vetch, winter field peas, lespedeza, alfalfa, cowpeas, nearly all commercial types of citrus, peaches, pecans, figs, strawberries, pears, bramble fruits, potatoes, onions, sweet potatoes, carrots, beans, spinach, table beets, cabbage, cauliflower, broccoli, turnips, watermelon, cantaloupes, squash, tomatoes, peppers, eggplant, and commercial greenhouse and nursery crops. The more serious diseases of a number of these crops have been the subject of continuous, intermittent, or brief terminating research studies by a considerable number of plant pathologists who have worked in Texas. To summarize their activities is the purpose of the following account.

Personnel

No plant pathologist appeared on the staff lists of the Texas Agricultural Experiment Station until 1909. Such studies of plant diseases as were made in the State before that were of brief duration, apparently conducted by the staff in other fields within the Station, or by men of external affiliations. L. H. Pammel, of St. Louis, Missouri, and Ames, Iowa, appears to have been the first of these. Between 1886 and 1889, he made the first technical examination of the destructive cotton root rot and demonstrated its cause to be a non-fruiting fungus which he identified as Ozonium auricomum. The fungus was later described as a new species, O. omnivorum, by C. L. Shear. Dr. Pammel's observations were published as Texas Agricultural Experiment Station Bulletins 4 and 7, in 1888 and 1889. T. L. Brunk, H. S. Jennings, and R. H. Price, horticulturists of the Station, left published accounts of plant diseases, chiefly of fruits, between 1889 and 1902. Price acquired the additional title of mycologist from 1899 to 1902. G. W. Curtis, director of the Station, wrote on alfalfa root rot, finding the cause to be the same as that earlier discovered by Pammel for the cotton disease. A bulletin on crowngall by H. Ness, botanist and horticulturist, was published by the Station in 1917.

F. D. Heald and F. A. Wolf, members of the botanical staff of the University of Texas from 1908 to 1912, made plant disease surveys in the region about San Antonio, publishing their observations in 1912. B. M. Duggar, of the Missouri Botanical Garden, made collecting trips into northeastern Texas in 1914 and 1915 and found the conidial stage of the cotton root rot fungus. Dr. Duggar's discovery led him in 1916 to transfer the organism to Bonorden's conidial genus, Phymatotrichum, as P. omnivorum, by which binomial the fungus is known today.

The subject of plant pathology was first accorded a staff position at the Texas Agricultural Experiment Station in 1909. R. H. Pond, the first incumbent, was succeeded in 1911 by F. H. Blodgett, who held the additional title of physiologist. When J. J. Taubenhaus came to College Station in 1916 as plant pathologist and physiologist, succeeding Blodgett, he found a largely untouched area from which to contribute phytopathological data. He applied a prodigious energy to

the gathering and reporting of information on all plant diseases which he observed within the State, to the adaptation of control measures known in older agricultural areas to crops under local conditions, and to special research problems requiring intensive, organized effort. In the latter category may be cited his long and varied studies on Phymatotrichum root rot, and his investigations on diseases of sweetpotatoes and onions. As the foundations grew Dr. Taubenhaus was able to add plant pathologists to his group until there were about twelve at the time of his death in 1937. From 1938 to 1950 the responsibility for leadership in phytopathology in the State was held by A. A. Dunlap. He continued the policy of aggressive attack on the more serious disease problems and performance of a great deal of diagnostic and other public service work as a means of maintaining current knowledge of plant disease conditions in the State. He increased the emphasis on plant physiological approaches to pathological and other problems of crop plants. Considerable effort was directed also to the development of locally adapted crops resistant to major diseases. During Dr. Dunlap's period of service the array of problems related to plant disease control in the State changed somewhat in response to the increased interest in organic compounds as fungicides, defoliants, weed-killers, and growth regulators.

Besides the pioneers and leaders mentioned above a considerable number of other investigators have taken part in plant disease research in the State. Their names, dates of phytopathological work in Texas, and the chief research interests of each are set forth in Table 1. This list includes not only plant pathologists, but certain agronomists, horticulturists, plant physiologists, biochemists, geneticists, etc., who have, in the opinion of the compilers, added importantly to the fund of knowledge of plant diseases in Texas.

Table 1. Persons who have taken part in plant disease research in Texas.

Name	Dates of activity	Research interests in plant pathology
L. H. Pammel	1886-1889	Phymatotrichum root rot
T. L. Brunk	1889	Grape diseases
H. S. Jennings	1890	General
R. H. Price	1892-1902	Fruit and vegetable diseases
G. W. Curtis	1892	Phymatotrichum root rot
H. Ness	1897-1929	Crown gall
F. D. Heald	1908-1912	General
F. A. Wolf	1908-1910	General
R. H. Pond	1909-1911	General
F. H. Blodgett	1911-1916	General
J. J. Taubenhaus	1916-1937	General, Phymatotrichum root rot, sweetpotato diseases, etc.
A. D. Johnson	1916	General
L. J. Pessin	1927-1928	General
B. F. Dana	1927-1931	Phymatotrichum root rot
W. J. Bach	1927-1935	Phymatotrichum root rot
J. P. Lusk	1927-1929	Phymatotrichum root rot
W. N. Ezekiel	1928-1944	Phymatotrichum root rot
G. W. Goldsmith	1928-1940	Phymatotrichum root rot, "live oak disease"
S. E. Wolff	1928-1934	Phymatotrichum root rot
P. R. Dawson	1928-1935	Phymatotrichum root rot
H. V. Jordan	1928-1942	Phymatotrichum root rot
E. R. Collins	1929-1936	Phymatotrichum root rot
I. M. Atkins	1930-	Diseases of small grains
Elizabeth Moore	1930-1949	Phymatotrichum root rot
Leta Henderson	1930-1936	Phymatotrichum root rot
D. R. Ergle	1930-1942; 1944-	Phymatotrichum root rot
S. E. Jones	1930-1950	Eggplant yellows
J. H. Hunter	1931-1940	Phymatotrichum root rot
C. H. Rogers	1931-1943	Phymatotrichum root rot, cotton seed treatments
D. C. Neal	1933-1936	Phymatotrichum root rot
N. E. Rigler	1934-1936; 1938-1943	Phymatotrichum root rot
G. T. Boyd	1934-1937	Rose diseases
L. B. Loring	1934-1935	Vegetable diseases

Table 1. -- (Continued)

Name	Date	Research interests
G. E. Altstatt	1934-1945	Tomato diseases
P. Decker	1934-1935	Phymatotrichum root rot
M. F. Kernkamp	1934-1935	Phymatotrichum root rot
L. E. Hessler	1934-1942	Phymatotrichum root rot
J. E. Adams	1935-1943	Phymatotrichum root rot
G. M. Watkins	1935-	Phymatotrichum root rot, rose diseases, peach diseases, southern blight
E. S. McFadden	1935-	Diseases of small grains
P. A. Young	1935-	Tomato diseases, general
D. C. Bain	1935-1937	Seed treatments
P. T. Riherd	1936-1937	Seed treatments
L. M. Blank	1936-1950	Phymatotrichum root rot, bacterial blight of cotton
G. A. Greathouse	1936-1939	Phymatotrichum root rot
E. C. Tullis	1936-	Rice diseases
G. H. Godfrey	1937-	Nematodes, Phymatotrichum root rot, citrus diseases, vegetable diseases, soil fumigation
A. L. Burkett	1937-1939	Seed treatments
E. W. Lyle	1937-	Rose diseases, Phymatotrichum root rot
S. S. Ivanoff	1937-1945	Spinach diseases, cantaloupe downy mildew
A. L. Harrison	1937-	Tomato diseases, watermelon diseases, peanut diseases
A. A. Dunlap	1938-1950	General, Phymatotrichum root rot, cotton physiology, diseases of ornamentals, 2,4-D injury
A. L. Martin	1938-1939	Rice diseases
H. Rich	1939-1940	Phymatotrichum root rot
C. Wilson	1939-1941	Phymatotrichum root rot
F. M. Eaton	1940-	Phymatotrichum root rot
C. E. Minarik	1940-1942	Rice diseases
G. KenKnight	1940-1942	Peanut diseases
F. E. Clark	1941-1944	Phymatotrichum root rot
R. B. Mitchell	1941-1944	Phymatotrichum root rot
R. J. Hervey	1942-	Phymatotrichum root rot
N. Higinbotham	1942-1944	Rice diseases
E. F. Paddock	1943-1944	Tomato diseases
S. M. Pady	1943-1944	Disease survey
H. W. Larsh	1943-1944	Disease survey
R. D. Watson	1943-1945	Rose diseases, disease survey
T. J. Nugent	1946	Spinach diseases
E. M. Hildebrand	1946-1949	Bacterial diseases
D. H. Bowman	1946-1948	Corn diseases
O. H. Calvert	1948-	Spinach diseases
L. S. Bird	1948-	Bacterial blight of cotton
M. D. Whitehead	1949-	Corn diseases
D. W. Rosberg	1949-	Pecan diseases, onion storage diseases
N. F. Clapp, Jr.	1949-	Rose diseases
H. T. Michael	1949-	Tomato diseases
Frances H. Brite	1950-	White tip of rice
H. R. Hudgins	1950-	Peanut kernel defects

Research

Through the decades since Dr. Pammel's observations in 1886-1889, the Phymatotrichum root rot of cotton and other crops has received the greatest sustained attention of all plant diseases known in the State. The studies of Pammel, Duggar, Taubenhaus, and many others resulted in a fund of detailed knowledge about the geographical occurrence of the organism in Texas,

its relation to climate and soil type, its very extensive host range and the losses inflicted on crops. C. H. Rogers, working at Temple, developed techniques and apparatus for determining numbers of sclerotia in large samples of soil, and conducted numerous studies relating sclerotial residues in the soil to various rotations, soil amendments and tillage practices. Data on various aspects of the nutrition of P. omnivorum were accumulated independently by C. H. Rogers, W. N. Ezekiel, and L. M. Blank. The processes by which the fungus enters the roots of cotton and other plants were examined by G. M. Watkins. Alkaloids and other constituents of root tissues as factors in the resistance of certain species were the subject of intensive investigation by G. A. Greathouse and N. E. Rigler. P. R. Dawson, J. E. Adams, L. E. Hessler, D. R. Ergle, H. V. Jordan and others at the U. S. Department of Agriculture Soil Fertility Laboratory at Austin, studied soil chemistry in relation to the prevalence and activity of P. omnivorum. G. W. Goldsmith, Elizabeth Moore, Leta Henderson, and others at the University of Texas devoted many years to various physiological and genetic aspects of cotton root rot. F. M. Eaton, N. E. Rigler, D. R. Ergle, and H. E. Joham have contributed extensive experimental data on the nutrition of the cotton plant and the level of carbohydrate reserves and other chemical factors within the plant in relation to cotton root rot. F. E. Clark, R. B. Mitchell and R. J. Hervey have analyzed the soil microflora in connection with various cultural practices designed to suppress the activity of P. omnivorum.

In the years following the discovery by C. J. King at Sacaton, Arizona, that Phymatotrichum root rot was strikingly controlled by very heavy applications of manure to infested soil, much attention was devoted to the development of a practical adaptation of Mr. King's principle suitable for use under conditions in Texas. The rather highly mechanized farming methods in use in the cotton areas of the State make manure a scarce commodity. From 1942 to 1946 E. W. Lyle, working at Temple, and L. M. Blank, conducting field experiments at various points in central Texas, demonstrated that the use of adapted winter legumes, planted in the fall and plowed under to decay a month before cotton planting time in the spring, would practically eliminate losses from Phymatotrichum root rot. The method has become standard farm practice in many parts of Texas.

Next to Phymatotrichum root rot bacterial blight (Xanthomonas malvacearum) is probably the most important cotton disease in the State. In many years serious midsummer defoliation results from the angular leaf spot phase of the disease. The work of C. H. Rogers at Temple showed that acid-delinting of seed, followed by treatment of organic mercury dusts, caused substantial reduction in seedling infection. Several years ago L. M. Blank initiated a program of introducing the resistance found in Simpson's Stoneville 20 into the major commercial varieties in use in Texas. The program, which has made use of standard plant breeding techniques, is being carried on by L. S. Bird, who has made studies of the inheritance of resistance. Considerable progress toward the development of blight-resistant strains of cotton has been made by D. R. Hooton and D. D. Porter, of the U. S. Cotton Field Station at Greenville.

For many years downy mildew (Pseudoperonospora cubensis) has been a major disease of commercial cantaloupes in south Texas. S. S. Ivanoff, working at Winter Haven, developed a new melon, released in 1945 as Texas Resistant No. 1, which possessed marked resistance to downy mildew and aphids. G. H. Godfrey, of the Lower Rio Grande Valley Experiment Station, has devoted several years to the development of new lines of cantaloupes based on crosses of good commercial types with a local wild cucurbit which is immune from downy mildew. Several of Dr. Godfrey's selections are presently in advanced tests at various locations in south Texas.

The so-called milo disease, caused by Periconia circinata, began to be observed as an important factor in the production of certain varieties of grain sorghum in northwest Texas about 1935. Although the pathogen was then unknown, R. E. Karper and J. R. Quinby, who observed the disease in connection with extensive sorghum breeding work going forward under their direction at Lubbock and Chillicothe, were able to select resistant sorghums and incorporated adequate resistance or tolerance into commercial varieties within a relatively short time.

An important commercial nursery area has existed for many years at Tyler, with rose bushes the principal product. E. W. Lyle conducted extensive experiments to develop adequate means of controlling black spot (Diplocarpon rosae), the chief disease of roses in the area. The system developed by Dr. Lyle of dusting nursery plants throughout the growing season with mixtures of sulfur and fixed copper compounds has been universally successful and is now standard practice among producers in east Texas.

Workers in the State have participated actively for many years in the coordinated regional efforts to combat rusts and other diseases of small grains through the development of resistant varieties. The leaders in this program within the State have been E. S. McFadden and G. W. Rivers at College Station and I. M. Atkins at Denton. Diseases of rice have been investigated at Beaumont since about 1936 by E. C. Tullis, A. L. Martin, C. E. Minarik and Noe Higinbotham.

Frances H. Brite is currently studying the use of fumigants as a means of preventing seed transmission of the nematode causing white tip.

The Texas Agricultural Experiment Station has entered recently into the southern regional program to conduct coordinated breeding work with corn, a portion of which involves resistance to major diseases. Research on corn diseases in Texas was initiated by D. H. Bowman and is being continued by M. D. Whitehead.

The lower Rio Grande Valley is an area characterized by intensive farming and an unusual diversity of crops. Plant diseases are ever present and often become limiting factors in production. G. H. Godfrey, who has served as plant pathologist at the experiment station at Weslaco since 1937, has confronted perhaps as great an array of plant disease problems there as ever faced any worker in the field. Although Dr. Godfrey's chief life-long research interest has been in phytopathogenic nematodes, the following partial list of his activities in Texas will suggest to some extent the breadth of every-day problems which have claimed his attention.

1. The discovery of Rio Grande gummosis of citrus, which he believes may be caused by an actinomycete.
2. Project leader of the current program to eliminate psorosis and other viruses from citrus by careful selection of budwood and maintenance of test nurseries.
3. Work with combination sprays and dusts, or "mist-dusts", for improving the adhesion of fungicides.
4. A study of stalk rot of corn, caused by Pythium butleri.
5. Experiments with new organic compounds for the control of late blight of potato.
6. Cultural control of root-knot nematodes by successively deeper plowings during hot, dry weather, especially applicable to short-season crops.
7. Development of a non-phytotoxic wound paint useful in surgery on citrus and other trees.
8. Use of nucleus colonies of bees for field selfing of cantaloupes under portable insect cages.
9. Correction of lime-induced chlorosis of citrus by means of manure composted with sulfur.
10. Identification of the citrus nematode in Texas.

E. O. Olsen is currently studying Phytophthora foot rot and other diseases of citrus nursery stock.

Important tomato-growing districts exist in several parts of the State, and for more than a decade the Agricultural Experiment Station has maintained two field laboratories devoted to research on diseases of the crop. One of these is operated by P. A. Young at Jacksonville and the other is under the direction of A. L. Harrison at Yoakum. Although the work of the two laboratories is marked by close liaison on problems of common interest, such as southern blight, Fusarium wilt, nematode root knot, and early blight, each is active in attacking purely local troubles. For example, during the past two years Dr. Young has experienced serious outbreaks of late blight in tomatoes in the vicinity of Jacksonville, while this disease has not occurred at Yoakum. Dr. Harrison's chief current interest is in producing strains of tomatoes resistant to Fusarium wilt, root knot, and the collar rot phase of early blight. He has developed inoculation methods that are particularly effective for the purpose.

Efforts to control scab and other diseases of pecan by means of copper sprays have been associated frequently with markedly increased aphid populations. Work was begun in 1949 by D. W. Rosberg to examine more closely the relationship between disease control and insect infestation in this case. The problem is complicated by a variety of factors, such as coverage problems in spraying large trees, great differences in scab susceptibility among host varieties, etc. The first complete season's results have, however, suggested the possibility of successful formulation of combined fungicides for future trials. Dr. Rosberg is studying also diseases of onions in storage in an effort to develop means of lengthening the marketing period.

The most important disease of spinach in southwest Texas is white rust (Albugo occidentalis). Efforts to develop practical control measures were made from 1937 to 1945 by S. S. Ivanoff and continued in 1946 by T. J. Nugent. Since 1948 O. H. Calvert has been experimenting with inoculation procedures for use in selecting for resistance and with altered crop management to permit the use of fungicides.

Commercial production of Spanish-type peanuts, which has become an important and highly organized activity in the State during the past decade, is hampered in most years by serious losses due to southern blight (Sclerotium rolfsii). In 1940 and 1941 Glenn KenKnight, working at Stockdale, grew a large number of peanut strains in heavily infested soils, seeking differences

in susceptibility to the disease. Such differences were observed. One of the least susceptible types was forwarded to B. C. Langley at Stephenville, who continued to work with the material until it was ready for release in 1948 under the name Spantex. B. C. Langley, A. L. Harrison, and G. M. Watkins are continuing the testing of new peanut seed lots for disease resistance and also are studying effects of certain cultural practices on losses inflicted by Sclerotium rolfsii.

From 1943 to 1945 the nation-wide Emergency Plant Disease Prevention Program was represented in Texas by G. E. Altstatt, S. M. Pady, H. W. Larsh, R. D. Watson, and G. M. Watkins. These men, none of whom was stationed in the State for the entire period, traveled almost constantly through the various farming districts and reported weekly to the Plant Disease Survey on the occurrence of crop diseases.

Plant pathologists in Texas are convinced that they are not likely soon to exhaust the array of crop disease problems requiring scrutiny. Occasionally a serious disease is introduced from another region, such as occurred when internal cork appeared recently in sweetpotatoes in east Texas. Plant breeders sometimes unwittingly combine excellent resistance to one major disease with spectacular susceptibility to an erstwhile minor trouble, leading to such difficulties as Victoria blight of oats. The rise of crop production costs has tended to narrow profit margins in many cases, with the result that disease losses formerly appearing negligible now loom increasingly large in the eyes of the growers. An important shift of interest toward a new class of crops brings not only new hosts but new disease problems. Such a thing is taking place now in Texas, where there is much enthusiasm for increased acreage in forage grasses and legumes. Plant pathologists in the State are at present greatly stimulated by an increased public awareness of their work and professional functions and by growing demands for their services.

TEXAS A. & M. COLLEGE SYSTEM, JACKSONVILLE AND COLLEGE STATION, TEXAS

THE ROLE OF PLANT PATHOLOGY IN VIRGINIA AGRICULTURE

S. A. Wingard

What has plant pathology meant to agriculture in Virginia? This, to me, is a very pertinent question and one whose answer is long past due. Taking stock of one's scientific accomplishments is most important and very revealing, yet it is something that most of us are prone to neglect. It is my purpose in the statement that follows to review briefly the history of plant pathology in Virginia with the hope that it may at least partially answer the question propounded above.

Plant pathology in Virginia, in comparison with other States, had a fairly early origin, yet it was too late to save the pear industry in southeastern Virginia, which reached the boom stage in 1880 and was suddenly snuffed out by the ravages of fire blight. The Virginia growers attributed the disease to over-heated sap, or to a surcharge of the electric fluid, and hung horseshoes in their pear trees to ward it off, although T. J. Burrill had already (1878) advanced the theory that fire blight was caused by bacteria. His theory was substantiated by later work (1881) of his own and completely confirmed by the experiments of J. C. Arthur in 1885. Despite this noteworthy discovery, control measures were not forthcoming and what promised to be a great Virginia pear industry died in its infancy.

Captain John Smith in 1629 reported that the colonists at Jamestown were able to produce peaches, apples, pears, apricots, vines, figs, and other fruits in abundance, although spraying for disease control did not come into existence until about 1887 following Millardet's discovery in 1882 that lime and copper could control downy mildew of grape. By 1892 Bordeaux spraying of grapes was common among growers in eastern Virginia, especially in Albemarle County. This practice, however, came too late to save the grape industry in that region, an industry that had its origin with the founding of the Jamestown Colony.

In the early years, the grape industry failed because the wine from the native grapes was too strong and heady; later because the introduced European grapes were so very susceptible to mildew and black rot. Finally, suitable varieties were developed by crossing the European and native varieties, and Bordeaux mixture was available for disease control, but by that time (1895) competition from California had increased to the point of discouraging Virginia growers. Thus, the grape industry was lost to Virginia, for periodic attempts since then to revive it have all failed. Nevertheless, grapes are still grown on a small scale for commercial and home use. Research on grape-disease control has continued and an experiment station bulletin on grape diseases and insects was published by R. H. Hurt in 1942. More recently research has been conducted with the newer organic fungicides, such as ferric dimethyl dithiocarbamate.

Apple, pear, quince, plum, peach, cherry, apricot and nectarine were introduced into Virginia from Europe, mainly as seeds, and planted by each early settler in the James River area as soon as he had succeeded in making clearings in the wilderness. These fruits were reported by Captain John Smith as being produced in abundance by 1629. Fruit growing was made compulsory by an act of the Virginia Assembly in 1639, but it remained almost wholly for home use up to 1854. Prior to this time orchard fruits were consumed mainly in liquid form or fed to hogs.

The improvement in transportation facilities between 1835 and 1887 paved the way for the commercial fruit industry of the State. Between 1860 and 1887 large plantings of peaches and pears were made on the south bank of the James River, in Surry County. These orchards proved unprofitable because the pear trees blighted and the fruit of the peach trees was destroyed at harvest time by the disease now known as brown rot. Practically nothing was known about fungus diseases at that time. A Virginia fruit grower is reported to have said in 1871 that "Rotting of peaches is due to an excessive flow of sap. If the growth of the trees is checked, by allowing grass to grow under them, a good crop is secured."

The winter apple industry had its birth about 1865, being made possible by the building of railroads into the Piedmont, the Valley, and Southwest Virginia, between 1840 and 1850. Impetus to the foreign demand for the Albemarle Pippin was brought about in 1837 by Andrew Stephenson, of Albemarle County, then Minister to the Court of St. James. A winter supply of home grown apples was shipped to him in London and he presented several barrels of Albemarle Pippins to Queen Victoria. She was so delighted with the flavor that she called for more from the same source and thus began the special demand for the Albemarle Pippin, as contrasted with the Newtown Pippin grown in the North, which later meant so much to the growers in Piedmont Virginia. It was pointed out by the editor of the Southern Planter in 1846 that the best fruit crop Virginia farmers could raise was the apple, the Pippin and some of the other fall and winter varieties being recommended. In 1877 growers in Albemarle County were shipping Pippins direct to Liverpool, England.

From the statement above, it would appear that the apple industry in Piedmont and Tidewater Virginia around 1880 was destined for a long period of prosperity. This, however, did not prove to be the case. The scene of pomological activity immediately shifted to other sections of the State, and the introduction of the refrigerator car in 1887 made it possible for distant fruit growers to compete with local markets. Diseases such as scab, bitter rot, cedar rust, and fire blight were taking a heavy toll. S. W. Fletcher, in 1932, had the following to say regarding the early apple industry in Virginia:

"The outlook for fruit growing in 1887 was not bright. The times were out of joint. Numerous gatherings of farmers resolved to 'view with great alarm the great depression which now rests upon our agricultural interests.' Prices were low. A horde of insect and fungus pests fattened on the trees, and no means of controlling them were known. 'The chief business of Virginia fruit growers in these days', lamented one of the growers, 'is to try to cheer one another up.' The apple industry, especially, was in the dumps."

Thus the apple industry, like the grape, the pear, and the peach industries, which preceded it, had suddenly come face to face with definite limiting factors, chief among which were fungus diseases (especially bitter rot) and insect pests. Hence, as a result of necessity, the fruit growers of Virginia turned to the sciences of plant pathology and entomology for relief. Fortunately for all concerned, the Virginia Agricultural and Mechanical College, now the Virginia Polytechnic Institute, had been chartered by the Assembly in 1872 and the Agricultural Experiment Station was established in 1888, thus making it possible for science to enter the field of fruit growing in Virginia.

It now seems nothing short of an act of Providence that William B. Alwood was appointed horticulturist and entomologist of the newly organized agricultural experiment station at Blacksburg in 1888, for it is to him, more than to any other, that the Virginia Fruit industry is indebted for its survival. To quote Fletcher again: "He stimulated and guided it while it was weak and despondent, devised and enforced laws for its protection, organized the State Horticultural Society, and showed qualities of leadership in many other directions which fruit growers are not likely to forget."

Dr. Alwood lost no time in developing the practice of spraying to help save the fruit industry. The first printed use of the term "spraying" in Virginia was in 1888, and was an account of experiments in Illinois in which Paris green was applied with a force pump to control codling moth. Bordeaux was used by Oscar Rierson of Glendover, Virginia, in 1887, for the control of grape rot. His results were not very favorable but others were more successful, and by 1892 Bordeaux spraying was common among grape growers, especially in Albemarle County. In 1889 Alwood contributed an article to the Southern Planter on "Apple Rusts", chiefly the cedar apple rust, and another on "Implements for Applying Insecticides and Fungicides," This, according to S. W. Fletcher, was the first scientific discussion of fungus diseases of fruit in Virginia.

The first crop pest law of Virginia, on peach yellows, was prepared by Alwood, and passed by the General Assembly in 1889. Without this foresight, peach yellows would probably have destroyed the peach industry. In 1892 he published the first full spray program for orchards in Virginia. The first spray calendar for disease and insects control was published two years later. Spraying with Bordeaux mixture saved the apple industry of Piedmont, Virginia, which at that time consisted chiefly of the Albemarle Pippin variety.

Alwood was a tireless worker and a prolific worker. He was working and publishing simultaneously in the fields of horticulture, entomology and plant pathology, yet his publications were of exceptionally high caliber and are so regarded even today. His publications on plant diseases had a momentous influence on the fruit industry of Virginia.

The Virginia fruit industry was indeed fortunate, after the retirement of Alwood to private life, to have his work in plant pathology continued by such able scientists as Howard S. Reed, E. B. Fred, J. S. Cooley, C. H. Crabill, H. E. Thomas, T. J. Murray, F. D. Fromme, and more recently by F. J. Schneiderhan, R. H. Hurt, and A. B. Groves. Virginia growers are fortunate also in being in close proximity to workers in the U. S. Department of Agriculture. W. M. Scott, C. L. Shear, M. B. Waite, Erwin F. Smith, Charles Brooks, D. F. Fisher, John W. Roberts and J. S. Cooley, of the Bureau of Plant Industry, deserve special mention for their contributions on the solution of fruit-disease problems in Virginia.

Many important fruit-disease bulletins have been published by research workers in Virginia who followed Alwood. In addition to these publications, members of the Experiment Station staff have published many timely articles on fruit-disease control in the "Southern Planter", "Virginia Fruit", and other agricultural magazines, as well as having presented papers before the State Horticultural Society and the American Phytopathological Society.

Research on fruit diseases and insects in Virginia has returned big dividends and the fruit industry of the State today owes its successful existence largely to the application of results of such work. Studies on cedar rust of apple resulted in the passage of the Cedar Rust Law by the

General Assembly in 1914, which enabled the fruit growers to eradicate red cedar trees in the vicinity of their apple orchards and thus control the destructive cedar rust disease. The commercial apple industry of the State, consisting primarily of plantings of rust-susceptible varieties, such as York, Rome Beauty, and Jonathan, would undoubtedly have been ruined had not this law been passed. Studies on other diseases and on spray materials for their control have resulted in the finding of practical methods for the control of most of these diseases.

Research plant pathologists are stationed at field laboratories in the major fruit-growing districts to study diseases and their control. Information thus obtained is passed on to the growers direct and through station bulletins, as well as through the bulletin "Information for Virginia Fruit Growers" which is revised annually; also, through the Spray Service of the Virginia Agricultural Extension Division, whereby each grower is not only notified when to spray but also what materials to use in each spray application.

It is of interest to note that in 1923 R. H. Hurt was employed by the Experiment Station and sent to Crozet to make a special study of fruit diseases in that section. He has continued to work in that section ever since and his research has been a major factor in the success of fruit growing in Piedmont Virginia. The fruit growers in the Piedmont section in 1930 expressed their appreciation for this work by donating $2,500 toward the construction of a laboratory building. The General Assembly then appropriated additional funds for its construction and equipment. The University of Virginia cooperated in the project by granting permission for the laboratory to be located on its grounds. This laboratory was maintained by the Virginia Agricultural Experiment Station for work in plant pathology and entomology until recently, when a modern laboratory building and a greenhouse, located near Charlottesville, were completed. R. H. Hurt is in charge of the research in plant pathology.

Fruit-disease research was begun at the Winchester Research Laboratory, Winchester, in 1922 by F. J. Schneiderhan who made many valuable contributions on apple scab, bitter rot, Missouri blotch, Xylaria root rot, and walnut toxicity. He was followed in 1929 by A. B. Groves who is still in charge of the fruit-disease research at that station. New buildings are now being constructed at the Winchester field station. A. B. Groves has made contributions on apple scab, sooty blotch, bitter rot, Xylaria root rot, measles, scald, cedar rust, fire blight, peach leaf curl, cherry leaf spot, sulfur fungicides, copper fungicides, new organic chemical fungicides, spray injury, and compatibilities of fungicides and insecticides on apples and cherries. His research at present includes long-time experiments on Xylaria root rot of apple, the evaluation of new fungicides and insecticides on apple scab, cedar rust, sooty blotch, peach curl, and the compatibility of new fungicides and insecticides.

Disease research has not been limited to fruit crops. Much attention has been devoted to research on the diseases of tobacco, peanuts, small grains, beans, tomatoes, cabbage, spinach, and other truck crops. Work is now underway on the diseases of apples, peaches, cherries, tobacco, tomatoes, beans, grapes, raspberries, small grains, corn, alfalfa, red clover, peanuts, soybeans, and other crops.

Tobacco has been a major money crop in Virginia since the colonists first settled at Jamestown. Tobacco in those days paid taxes to the mother country, and two hundred pounds of tobacco paid the fare of an English lass, who later became the wife of a pioneer bachelor. Tobacco is troubled with many diseases: leaf spots, mosaic, ring spot, Granville wilt, blue mold (downy mildew) sore shin, southern blight, root rot, and, more recently, the most destructive of all, black shank. These diseases have commanded the attention of research workers for many years. Investigators of the Experiment Station at Blacksburg, the several branch experiment stations, and the U. S. Department of Agriculture have made valuable contributions in the solution of these tobacco-disease problems.

Two bacterial diseases of tobacco, wildfire and angular leaf spot, appeared in Virginia in 1918 and caused very severe losses for several years. F. D. Fromme, S. A. Wingard, and T. J. Murray did pioneer work on these diseases and they were eventually brought under control. Ring spot of tobacco received attention from F. D. Fromme, S. A. Wingard, C. N. Priode, and R. G. Henderson. Tobacco mosaic and black root rot have been a special project of R. G. Henderson for several years. He has succeeded in developing good commercial varieties of tobacco of different types with resistance to these diseases. Blue mold of tobacco appeared in Virginia in 1932 and for several years caused great damage to tobacco seedlings in plant beds. Pioneer work on this disease by J. A. Pinckard and R. G. Henderson in cooperation with workers in the U. S. Department of Agriculture, Duke University, and other State experiment stations resulted in practical control measures. G. M. Shear made valuable contributions on the physiological diseases of tobacco commonly known as frenching and mature leaf breakdown.

A tobacco disease research station was established at Chatham in 1936 with J. A. Pinckard in charge. He studied tobacco mosaic, black root rot and blue mold and made contributions on

all three diseases. He was followed in 1941 by W. A. Jenkins, who has devoted the greater part of his time to studies on meadow nematode and black shank of tobacco on which he has made invaluable contributions. Black shank appeared in Virginia in 1937 and spread rapidly over the State. Many farms were rendered unfit for tobacco production by black shank infestation until Jenkins developed good resistant varieties which are now being grown on thousands of acres in Virginia and North Carolina. New strains of tobacco with combined resistance to black shank, Granville wilt, mosaic and root rot are now being developed.

A 78-acre farm near Chatham was purchased by the State in 1948 for growing seed of the black shank-resistant varieties for commercial use, and for tobacco disease research, especially the breeding of disease-resistant varieties. W. A. Jenkins is in charge of the disease research and J. L. LaPrade of the seed production. Cooperative work is being done on the development of black shank-resistant burley tobacco varieties with workers in Kentucky and Tennessee.

Prior to 1947 research in Virginia on the diseases of corn and small grains was limited to minor work by Fromme, Wingard, and Shear on the stalk and ear rots of corn, nutritional diseases and seed treatment of corn, seed treatment of small grains, nematode and black stem rust of wheat, and breeding for loose smut-resistant varieties of wheat. Beginning in 1947 Curtis W. Roane has devoted full time to research on the diseases of corn and small grains and G. M. Shear has continued his studies on some of the deficiency diseases.

Roane is working cooperatively with the plant breeders in the Agronomy Department of this experiment station and the U. S. Department of Agriculture in an effort to develop disease-resistant varieties of small grains and corn hybrids. The corn diseases receiving major emphasis are the leaf blights (Helminthosporium turcicum, H. maydis, and H. carbonum), and stalk rots (Gibberella fujikuroi, G. zeae, and Diplodia zeae). An attempt is being made to develop barley with resistance to leaf rust, loose smut and powdery mildew; oats with resistance to crown rust, Victoria blight, loose smut and stem rust; and wheat with resistance to leaf and stem rust, powdery mildew and loose smut.

R. G. Henderson, in cooperation with T. J. Smith and others, is attempting to develop disease-resistant strains of alfalfa adapted to Virginia conditions. Diseases caused by the following organisms are receiving major consideration: Sclerotinia trifoliorum, Corynebacterium insidiosum, Ascochyta imperfecta, Colletotrichum trifolii, Pseudopeziza medicaginis, Stemphylium botryosum, Stagonospora meliloti, Rhizoctonia sp., and Ditylenchus dipsaci. Stem nematode was discovered in Virginia for the first time in 1948 near Richmond and is apparently still restricted to that one location. The infested area has been taken over for experimental work. Several of Oliver F. Smith's nematode-resistant strains of alfalfa are being tested on this area and some of them show considerable promise.

Research on peanuts by L. I. Miller , S. A. Wingard and E. T. Batten has dealt primarily with chemical seed treatment, sulfur and copper dusting for leaf spot control, and Sclerotium wilt. Seed treatment has greatly improved the stand of plants and enabled the peanut growers to use machine-shelled seed, which means a great saving over hand-shelled seed. At least 99 percent of the growers now practice seed treatment. Sulfur dusting for leaf spot control is generally practiced and it has greatly increased the yield and quality of both hay and nuts.

G. M. Shear and L. I. Miller have made some valuable contributions on manganese deficiency and overliming of peanuts. They are continuing their studies on these and other nutritional problems of peanuts.

Work on the diseases of vegetable crops has been conducted both at the Virginia Agricultural Experiment Station, Blacksburg, and the Virginia Truck Experiment Station, Norfolk. The work at Blacksburg has dealt with studies by Fromme on the development of cabbage resistant to yellows, studies on bean and cowpea rusts by Fromme and Wingard, the development of rust-resistant varieties of snap beans by Wingard, experiments on club root of cabbage by H. S. Reed, yeast-spot of lima bean by Wingard, bacterial soft rot of tomato by Wingard and Massey, and the control of the leaf blights and fruit rots of tomatoes by Reed, Thomas, Fromme, Wingard, and Henderson.

The region around Norfolk has long been noted for its fine truck crops, an industry that owes its present existence to research conducted in plant pathology, horticulture, and entomology by the Virginia Truck Experiment Station. The establishment of the Truck Experiment Station was made possible by the Southern Produce Company, a cooperative vegetable marketing and shipping organization, which in 1907 donated 57 acres of land for a truck experiment station and also provided the necessary buildings and equipment. From 1907 to 1920, funds for operation were furnished from the fertilizer sales tax fund of the State Department of Agriculture at Richmond and by the sale of produce at the Truck Station. In 1920 the Virginia Truck Experiment Station was established as a permanent State institution by an Act of the General Assembly and given

adequate financial support. The research staff has been greatly increased in recent years and subsequent purchases and leases of land have expanded the experimental area.

During the early days of the Truck Experiment Station, the pathological work was handled by L. L. Harter of the U. S. Department of Agriculture, who devoted most of his attention to certain malnutrition diseases of truck crops, especially of cabbage and spinach. He made important contributions on the solution of these problems. During this period T. C. Johnson, then director, also contributed several publications on potato diseases.

The discovery of the true nature of spinach blight (mosaic) by J. A. McClintock and L. B. Smith and the development of the resistant variety, Virginia Savoy, by McClintock, Smith and H. H. Zimmerley and the resistant variety, Old Dominion, by Zimmerley were contributions of great importance in our knowledge of virus diseases and the control of diseases by the breeding of resistant varieties. This work undoubtedly saved Norfolk as a spinach growing area.

From 1920 to 1924, Zimmerley, F. W. Geise, Herbert Spencer and R. J. Davis made important contributions on the dusting and spraying of various truck crops for the control of insects and diseases. F. P. McWhorter made important contributions on the treatment of beet seed for the control of seedling diseases and on the Fusarium wilt and early blight diseases of tomatoes. He also cooperated with the U. S. Department of Agriculture in investigations on the diseases of certain ornamentals, especially of narcissus and poppy.

In 1931 and 1932, Harold T. Cook published the first extensive papers on the powdery mildew disease of snap beans and methods of control. He also published extensively on vegetable seed treatment. This research showed that treating spinach seed with red copper oxide would insure a satisfactory stand of spinach, whereas formerly the growers had had much difficulty in obtaining satisfactory stands. Later experiments showed that as good results could be obtained by treating with zinc oxide, which costs less and is less irritating to the respiratory passages of the operators. The annual saving in cost of treating spinach seed by substituting zinc oxide for red copper oxide is considerable for the Norfolk area alone. The gain in crop value from treating spinach seed often amounts to more than $100 an acre, while the cost of treating the seed is only about 15 cents an acre. In the Norfolk area it has now become a regular practice for the seedsmen to treat all spinach seed for the growers at about cost.

Investigations by Cook also resulted in finding a wilt-resistant variety of watermelon, the Hawkesburg, which is similar in shape and color to the Owens Grey variety commonly grown in the Smithfield area where the wilt disease made it unprofitable to grow the ordinary varieties on many of the farms. Cook and Nugent investigated the effect of acid-forming and non-acid forming fertilizers on the development of potato scab and obtained evidence that the disease was influenced only to the extent that the fertilizers changed the soil reaction. This reassured the farmers that it was safe to use the more desirable non-acid forming fertilizers as long as the soil reaction was kept in the neighborhood of pH 4.8 which was unfavorable for scab development.

In 1940 Cook and Nugent published a manual entitled "The Control of Truck Crop Diseases in Tidewater Virginia". This manual was written in a new style in which the control measures were given for the disease of the crop as a whole rather than for individual diseases. This arrangement made the manual much more practical for the use of county agents, agricultural teachers, and farmers. A publication was prepared by Cook and Harter on the results of tests of new fungicides for treating seed sweetpotatoes to meet the new emergency brought about by the wartime scarcity of the fungicides formerly used for that purpose. Some of the substitutes discovered in these investigations have become permanent because they are more desirable in a number of ways than the materials formerly used. The vegetable disease research at the Virginia Truck Experiment Station is now in the hands of R. S. Mullin and T. J. Nugent.

The work in plant pathology at the Virginia Ploytechnic Institute, as pointed out above, was begun in 1888 by William B. Alwood, then horticulturist and entomologist, of the experiment station. A Department of Bacteriology and Plant Pathology was organized in 1908 with the late Howard S. Reed as head. Reed was succeeded by F. D. Fromme in 1916, and he in turn by S. A. Wingard in 1928. Botany was transferred in 1922 from the Department of Botany and Zoology to the Department of Bacteriology and Plant Pathology and the name of the latter changed to Department of Botany and Plant Pathology. In 1935 the Department of Botany and Plant Pathology was combined with the Department of Entomology and the Department of Zoology and Animal Pathology to form the Biology Department. On September 1, 1949, plant pathology and physiology were separated from the Biology Department and set up as a Department of Plant Pathology and Physiology with S. A. Wingard again as head.

At present there are six men in the department (A. B. Groves, R. H. Hurt, W. A. Jenkins, L. I. Miller, J. L. LaPrade, and Luben Spasoff) who devote full time to research in plant pathology and five (S. A. Wingard, R. G. Henderson, G. M. Shear, C. W. Roane, and J. I.

Shafer, Jr.) who divide their time between research and teaching in plant pathology and physiology. S. B. Fenne and J. M. Amos handle the extension work in plant pathology.

VIRGINIA POLYTECHNIC INSTITUTE, BLACKSBURG

PLANT PATHOLOGY IN THE STATE OF WASHINGTON, -- PAST, PRESENT, FUTURE

George W. Fischer [1]

INTRODUCTION

Washington is a large and climatically diverse State. East of the Cascade Range the average annual precipitation varies from seven inches in the central part of the State to nineteen in the eastern; west of the Cascades, from forty to one hundred and twenty inches. In eastern Washington dry land farming predominates, with wheat (and to a lesser extent other cereals), dry edible peas, alfalfa, and other forage crops forming the major crops. The intense bright sunshine in the central part of the State makes this region especially adapted to the production of quality tree fruits for which Washington is so well-known. These tree fruits are dependent on irrigation, as are in fact the great variety of vegetable and field crops for which the area is also acquiring a reputation. West of the Cascade Range, agriculture, is similarly diversified to include strawberries, bramble fruits, tree fruits, ornamental bulbs, cranberries, blueberries, walnuts, filberts, peppermint, cereals, potatoes, squash, cabbage, beets, snap beans, sweet corn, and other vegetables, vegetable seed production, and abundant pasture land, besides the vast forested areas for which the State is indeed well-known. In western Washington and to a lesser extent in parts of eastern Washington are the extensive evergreen forests which give Washington its name, "The Evergreen State." Since the diversity of the climate and topography of the State have given rise to such diversity of crops, it can readily be appreciated that there must follow a diversity also of production problems. Plant diseases have been prominent among these, and have provided a perpetual challenge to the plant pathologists and to the plant breeders as well.

HISTORICAL ACCOUNT 1892-1950

The early development of plant pathology in the State of Washington is inextricably woven around the life and activities of one outstanding plant pathologist, -- F. D. Heald. Before Dr. Heald's advent to the State College of Washington, there was no such thing in that institution as a Department of Plant Pathology. The State College of Washington was established in 1890, and for many years Botany, Zoology (including Entomology), and Bacteriology were combined into one department. For the first few years C. V. Piper was professor of the Department and apparently its sole member. In the Experiment Station Staff of six, Piper was also "Botanist and Entomologist." Professor Piper, in the 1893 annual report of the Experiment Station, said that the great prevalance of wheat smut is the most important production factor and "all is due to carelessness in treating seed."

The nearest approach to a course in plant pathology came in 1893 with a course in "Cryptogamic Botany." The description given in the Third Annual Catalogue (1893-94) describes this course as follows: "An introduction to the morphology and classification of fungi. Special attention will be given to economic forms, the means of controlling them, and methods of tracing life histories. Two days in the week throughout the year." Professor Piper complained that, owing to the press of college duties, he could not give adequate attention to the many problems of plant diseases and insect pests. Specifically mentioned, besides the all-important wheat bunt, were peach mildew, peach leaf curl, mildews of many crops, tomato blight (now known as curly top), crown gall of grape and apple (the latter widespread in eastern Washington). Orange rust of raspberry was introduced into Washington in 1895 and Professor Piper recorded an attempt to eradicate this disease. (Apparently the attempt was unsuccessful, or the disease was reintroduced later, because orange rust is commonly found in western Washington.) From this embryonic beginning, we can trace the development of plant pathology through the succeeding years.

In 1896, R. W. Doane was appointed to the Department of Botany, Zoology, and Bacteriology, and a course in "Systematic Botany" was added to the curriculum. The catalogue for that year stated that for the latter course "The fungi and bacteria receive most attention during the first semester."

In 1902, when the 11th Annual Catalogue was issued, C. V. Piper was still listed as Professor, but with added staff members as follows: Robert E. Snodgrass, Assistant Professor of Entomology; R. Kent Beattie, Instructor in Botany, and Assistant Botanist in the experiment station; and Herbert S. Davis, Instructor in Zoology. Two plant pathologsist were temporarily

[1]The author gratefully acknowledges the help of several of his colleagues in checking the manuscript, and furnishing suggestions for improvement.

stationed at the Western Washington Experiment Station at Puyallup: David A. Brodie (Superintendent), and Wm. H. Lawrence. Brodie worked on potato late blight control and Lawrence on blackberry crown gall, among many other problems.

In 1903, C. V. Piper resigned to take a position with the U. S. Department of Agriculture, and in the 1904 W.S.C. catalogue, R. Kent Beattie was listed as "Acting Professor of Botany." A. L. Melander was added as "Instructor in Entomology." In the 1903 Experiment Station Annual Report, Professor Piper indicated that root rots of orchard trees continued to be bad in Clark County. The cause was determined as Armillaria mellea. This was described in popular Bulletin No. 59 "Root diseases of fruit and other trees caused by toadstools", by C. V. Piper. It was further recorded in the 1903 Experiment Station Annual Report that in spite of the general use of blue vitriol, very large annual losses continued due to smut diseases of grain. In the same year an attempt apparently was made to control the Mormon cricket by use of "The South African Grasshopper Fungus." The attempt was totally ineffective.

In 1904 William H. Lawrence apparently completed his assignment at Puyallup and was returned to the Pullman Station as "Assistant in Botanical Investigations." In the 1904 Annual Experiment Station Report, Professor Piper stated that tremendous losses were being suffered due to smut in wheat and oats and to threshing machine explosions, resulting from threshing very smutty grain. The incidence of wheat smut ran as high as 40 percent in single fields.

In 1905, two new courses in instruction were added:

"3. Morphology and Classification of Fungi" (the old course in Cryptogamic Botany).
"9. Economic Fungi."

The course description for "Economic Fungi" as given in the 1905 catalogue was as follows:

"A study of the more common plant diseases due to fungi; their distribution, the life histories of the parasites; culture methods and methods of combatting them. Two lectures and 6 hours laboratory per week, first semester, Mr. Lawrence."

This new course is, as far as I can determine, the first bona fide course in plant pathology although it is not called by that name. William H. Lawrence was the instructor.

In 1907, Zoology was removed to become an independent Department, leaving a Department of Botany and Bacteriology of which R. Kent Beattie was professor, and William H. Lawrence and Samuel W. Collett instructors.

In 1908, William H. Lawrence resigned to take charge of the newly created Western Washington Experiment Station at Puyallup and also to serve as Plant Pathologist at that Station. Apparently this was the first instance where an experiment station man in the State of Washington received the title of Plant Pathologist or any subrank thereof. In 1909, a now very familar name appeared in the College catalogue, -- Harry B. Humphrey, as Assistant Professor. The course "Fungi" was split into two five-hour sequence courses while the course "Economic Fungi" was unchanged. All three courses were taught by Professor Humphrey. Another now well-known botanist and plant pathologist was added to the staff in 1909 with the appointment of N. Rex Hunt.

In 1911 came the first designation in the State College of Washington of any professorial rank in plant pathology, when H. B. Humphrey was listed as "Professor of Plant Pathology." Dr. Humphrey taught courses in Fungi, Cytology, Algae, Ecology, Histology, Bacteriology, and Marine Algae in addition to helping with one other course. It would appear that Dr. Humphrey was a busy man in those days. Also in 1911 William H. Lawrence resigned and was replaced by another plant pathologist who has since become well-known in his field -- H. P. Barss.

The department of Botany in the Experiment Station was abolished in 1911 and in its stead there was established a Department of Plant Pathology in the Experiment Station, with H. B. Humphrey as Plant Pathologist in charge.

What is possibly the nucleus of the eventual segregation of Plant Pathology from its mother science, Botany, came about in 1912 when the Botany courses were divided among sections: Morphology and General Botany; Physiology and Ecology; Pathology; Technical Courses. Under "Pathology" we find 4 new or newly designated courses:

"51. General Plant Pathology. 3 hours. Introductory Course.
"52. Plant Pathology. 3 hours. Morphology and Systematics of Pathogenic Bacteria, Slime molds, and Phycomycetes.
"53. Plant Pathology. 4 hours. Continuation of 52 to include Ascomycetes, Basidiomycetes, and Fungi Imperfecti.
"54. Forest Fungi. 2 hours."

It is thus seen that by 1912, and probably largely through Professor Humphrey's influence, the instruction in Mycology and Plant Pathology had begun to take quite a definite form. It must be remembered that in addition to Professor Humphrey's teaching activities, he served as Plant Pathologist and Vice Director in the Experiment Station from 1910 to 1913. Further evidence of Professor Humphrey's successful activities is seen in the 1912 Agricultural Experiment Station Report with the statement "The Division of Plant Pathology is now fairly well-equipped as to laboratory facilities, microscopes, and other necessary apparatus; and it is now possible to push to completion one by one the several projects before us."

In 1912, H. P. Barss resigned to take a position with Oregon State College and Experiment Station, and H. B. Humphrey left in 1913 to head up the pathology activities of the Office of Cereal Investigations in the Bureau of Plant Industry at Washington, D. C. He was succeeded by Ira D. Cardiff as "Professor and Head of the Department and Plant Physiologist in the Experiment Station." In the same year another reorganization took place, probably as a result of the change in headship. The Division of Plant Pathology was abolished and in its stead was organized a Division of Botany in the Experiment Station, with Cardiff as head. John G. Hall was added to the staff as "Professor of Plant Pathology and Plant Pathologist in the Experiment Station," and D. C. George as "Instructor and Assistant Plant Pathologist." This it is seen that by 1914 there had been established a definite concept of dual responsibility in teaching and research vested in the same individual rather than assigned to separate individuals.

In 1913 the United States Department of Agriculture established a field laboratory and station at Wenatchee in support of investigations of tree-fruit crops in that area. Pathological investigations were conducted by D. F. Fisher. Fisher was active in investigations of storage decays, apple mildew, and other fruit diseases.

In 1914, H. M. Woolman was added as "Assistant in Plant Pathology." This latter title is equivalent to the present rank of "Junior Plant Pathologist."

In 1915, there arrived the man who was to have more influence on the welfare of the Department that any other person. F. D. Heald was appointed as "Professor of Plant Pathology and Plant Pathologist in the Experiment Station." He succeeded Hall, who resigned. In the same year, two new courses of instruction were added:

"Botany 55: Diseases of Fruits.
"Botany 56: Methods in Plant Pathology."

The roster of courses in Plant Pathology was expanded during the following two years, and in the 1917 catalogue under the section "Plant Pathology" of the Department of Botany and Bacteriology, there were listed:

"P. P. 1. General Pathology; 5 hours.
P. P. 2. Crop Diseases; 3 hours.
P. P. 11. Fungi of Plant Disease; 3 hours.
P. P. 12. Methods in Plant Pathology; 3 hours.
P. P. 13. Principles and Practices of Disease Control; 3 hours.
P. P. 16. Forest Pathology; 2 hours.
P. P. 19. Diseases of Fruits; 3 hours.
P. P. 21. Diseases of Cereal and Forage Crops; 2 hours.
P. P. 40. Problems in Plant Pathology.
P. P. 41. Research in Plant Pathology."

The 1917 catalogue indicated that all these courses were taught by Dr. Heald. At the same time he was Plant Pathologist in the Experiment Station. Doubt our story if you can, but F. D. Heald was a busy man. How any man could teach all these courses and serve the demands involved as Plant Pathologist in the Experiment Station is something that is very hard for most of us today to comprehend. At the same time he gave tremendous impetus to the investigations of wheat smut.

The year 1918 was outstanding in the history of the Department of Plant Pathology at the State College of Washington, because it was in that year that the department itself was elevated to a full-fledged independent Department of Plant Pathology. At the same time the research Division of Plant Pathology was re-established in the Experiment Station. In the 1918 catalogue, we find for the first time a "College of Agriculture." Dr. F. D. Heald was head of the newly created Department of Plant Pathology in the College of Agriculture and of the Division of Plant Pathology in the Experiment Station, which position he retained until his retirement in 1941.

Also the year 1918 saw the advent of the first Extension Plant Pathologist in the State of Washington, in the person of A. M. Christensen. He stayed but a short time and was succeeded

by Noel F. Thompson, who also soon left and was promptly succeeded by George L. Zundel. Judging from the available records, all these changes took place during the year 1918.

Also in 1918, B. F. Dana was added to the staff of the department of Plant Pathology as Instructor and Assistant Plant Pathologist to replace Woolman, who resigned to accept a position with the Office of Cereal Investigations, Bureau of Plant Industry. Of interest now is the record in the 1918 Experiment Station Annual Report of the first report from the State of Washington of the aecial stage of stem rust on the common barberry.

In 1919 two new courses were offered in Plant Pathology. They were "P. P. 15, Diseases of Truck Crops", and "P. P. 42, Seminar." In the same year, Edward C. Johnson, a Cereal Pathologist in his own right with the U. S. Department of Agriculture, assumed his duties as Dean of the College of Agriculture and Director of the Agricultural Experiment Station. In 1923 D. J. Crowley was appointed in charge of the cranberry laboratory at Long Beach to conduct investigations on diseases and insects of cranberries in that region.

No further changes in staff or instruction took place until 1925 when E. E. Honey was added to the staff as Instructor in Plant Pathology. Apparently Dr. Honey was a young, unusually enthusiastic collector, not just for pathological and mycological items but for many other things of interest to him as evidenced by a record which Dr. Heald left in his office and which is typical of the latter's wit: "Bees collect honey but Honey collects".

The year 1926 is another landmark in the history of the Department for it was in that year that Dr. Heald's "Manual of Plant Diseases" was published. The place that this book found in the science of Plant Pathology all over the world is well-known and needs no elaboration here. Also in 1926, Dr. Zundel resigned as Extension Assistant Professor in Plant Pathology to go to the Connecticut Experiment Station to work with G. P. Clinton.

In 1926 Honey resigned to take a position with the New York State Extension Service. He was replaced by George D. Ruehle as Assistant in Plant Pathology. In the same year the courses of instruction were renumbered so that they appeared in the catalogue as follows:

"P. P. 101 General Pathology
P. P. 110 Fungi of Plant Disease
P. P. 111 Fungi of Plant Disease (2nd Sem.)
P. P. 112 Methods in Plant Pathology
P. P. 113 Principles and Practices of Disease Control
P. P. 115 Diseases of Truck Crops
P. P. 116 Forest Pathology
P. P. 119 Diseases of Fruits
P. P. 121 Diseases of Cereal and Forage Crops
P. P. 140 Problems in Plant Pathology
P. P. 142 Seminar
P. P. 241 Research in Plant Pathology"

Several of these courses were offered in alternate years only.

Plant Pathology in the western part of the State received some support in 1927 with the appointment of G. A. Newton as Plant Pathologist at the Western Washington Experiment Station at Puyallup.

In 1927, Dana resigned to accept appointment as plant pathologist at the Texas Agricultural Experiment Station. He was replaced by Lee E. Miles as Assistant Professor of Plant Pathology Also in 1927, E. L. Reeves was appointed by the U. S. Bureau of Plant Industry as Field Assistant in the Division of Fruit and Vegetable Crops and Diseases and stationed at Wenatchee to work on diseases of tree fruits.

Miles resigned in 1928 and was succeeded by Leon K. Jones as Assistant Professor and Assistant Plant Pathologist in the Experiment Station. H. H. Flor was added to the staff as Associate Pathologist, U. S. Department of Agriculture, to work with Dr. Heald on the wheat smut problem. He was the first Federal Plant Pathologist to be appointed to the Washington Agricultural Experiment Station.

In 1930, the first doctorate in Plant Pathology was awarded. The recipient of this honor was George D. Ruehle. Incidentally, this was the first doctorate to be awarded from the entire institution. Following his graduation in 1930, Ruehle resigned his position as Assistant in Plant Pathology to accept appointment with the Citrus Experiment Station in Florida. In 1930, three courses were added to the Plant Pathology curriculum: "P. P. 210 and 211, Advanced Mycology" and "P. P. 117, Diseases of Ornamentals." Also in 1930, W. D. Courtney was appointed by the U. S. Bureau of Plant Industry as Junior Nematologist at the Western Washington Agricultural Experiment Station to begin investigations of nematode troubles in Washington and Oregon.

In 1931, C. S. Holton was added to the staff as agent, U. S. Department of Agriculture, to replace Dr. Flor who was transferred to Fargo, North Dakota.

No further changes took place until 1932 when Glen A. Huber was added to the staff as "Instructor and Assistant in Plant Pathology." Huber taught the beginning Mycology courses (P. P. 110 and 111), Methods in Plant Pathology (P. P. 112), and with Jones, General Plant Pathology (P. P. 101). Also in 1932 the second edition of Heald's Manual appeared.

In 1934, Dr. Huber resigned to take a position as Plant Pathologist at the Western Washington Experiment Station in Puyallup. He was replaced by George W. Fischer as half-time Instructor in Plant Pathology, teaching the two Mycology courses, the methods course, Forest Pathology, and the beginning course with Jones, and the Advanced Mycology course with Heald.

To further augment plant disease investigations in western Washington Leo Campbell was assigned in 1935 to work on vegetable diseases in the northwest corner of the State. He was principally concerned with diseases of beans and beets, but at the same time included other vegetables and also began a period of investigations on strawberry diseases which was to continue for many years.

In 1936, Fischer resigned to accept appointment with the U. S. Bureau of Plant Industry to work on diseases of forage grasses. His headquarters remained at Pullman. W. Harley English was appointed as half-time Instructor in Plant Pathology to replace Fischer.

In 1937, Professor Heald was granted a year's leave of absence and was gone from the Department from July 1937 until June 1938. S. M. Dietz was imported from Iowa State College to serve as Acting Head of the Department. Dr. Campbell was transferred to the Western Washington Experiment Station as Assistant Plant Pathologist.

Plant Pathology work was first started at the Irrigation Experiment Station at Prosser when, in 1938, Richard Wellman was appointed to that Station as Junior Plant Pathologist. He left the following year to take a research position with Boyce Thompson Institute and was replaced by J. D. Menzies, who began a research program emphasizing the diseases of alfalfa and virus diseases of stone fruits, particularly in the Yakima Valley.

In 1939, English resigned to accept appointment with the U. S. Department of Agriculture at Wenatchee, to work on problems of decay of apples and pears in storage and in transit. D. M. Coe succeeded him as Instructor in Plant Pathology.

No further changes in personnel or instruction took place until 1941 when Heald was retired as Emeritus Professor. He was succeeded by J. G. Harrar as Professor and Head of the Department and Chairman of the Division of Plant Pathology in the Experiment Station. Also in 1941 C. J. Gould was appointed Assistant Plant Pathologist at the Western Washington Experiment Station to begin some badly needed investigations of diseases of ornamental crops, particularly bulbs.

Another important phase of the plant pathology program in the State of Washington was begun in 1942 with the establishment, by the State Department of Agriculture, of the Plant Introduction and Quarantine Station, at Moxee. D. M. Coe was the first plant pathologist in charge of the Station. He collaborated with Reeves in extensive surveys of stone fruit viruses in central Washington and in laying much of the ground work for the future functioning and importance of the Station.

In 1943, Harrar resigned to accept a position with the Rockefeller Foundation in Mexico. E. J. Anderson was appointed to succeed Harrar. Also in 1943 Folke Johnson was appointed at the Western Washington Experiment Station as Assistant Plant Pathologist to work on certain diseases of tree fruits and cane fruits.

In 1944, the Division of Fruit and Vegetable Crops and Diseases, U. S. Bureau of Plant Industry, appointed Glen Pound as Assistant Pathologist at the Northwest Washington Experiment Station at Mount Vernon, to work on the more important diseases affecting the vegetable seed crops in that area. L. K. Jones resigned to accept a position with the U. S. Bureau of Entomology and Plant Quarantine. In the same year, Washington became the 18th State to join the Federal Government in the campaign to eradicate barberry bushes. Mr. H. B. Busdicker was appointed as State leader and Plant Pathologist, U. S. Bureau of Entomology and Plant Quarantine.

In 1945, Anderson resigned to accept a position with the Pineapple Research Institute in Hawaii. Fischer resigned his position as Pathologist with the Division of Forage Crops and Diseases, U. S. Bureau of Plant Industry at Pullman, to accept appointment with the State College of Washington, replacing Anderson. Earlier in the same year S. B. Locke was added to the staff as Assistant Professor and Associate Plant Pathologist. Also in 1945, Menzies resigned his position with the Irrigation Experiment Station to accept appointment as Pathologist with the U. S. Department of Agriculture, Columbia Basin Investigations Project but maintained headquarters at the Prosser Station. This project was begun to furnish research information on the

vast Columbia Basin Irrigation Project prior to the actual settlement of the area.

In 1946 several changes took place. George Nyland was added to the Plant Pathology staff as Instructor and Junior Pathologist in the Experiment Station. D. M. McLean was appointed as Associate Pathologist, U. S. Department of Agriculture, to succeed Glen Pound at the Mount Vernon Station. Dr. Pound had resigned earlier in the year to go with the University of Wisconsin. Frank V. Stevenson was appointed later in the same year as Assistant Professor of Plant Pathology and Assistant Pathologist in the Experiment Station. M. R. Harris was appointed as Extension Plant Pathologist; he was the first extension plant pathologist to be appointed since Dr. Zundel resigned in 1926. Finally, in 1946, an important step took place when E. C. Blodgett was appointed jointly with the Washington State Department of Agriculture as Associate Plant Pathologist to head up a program of research, survey and nursery stock improvement in the field of stone fruit viruses, and to supervise the work at the Plant Introduction and Quarantine Station at Moxee.

The year 1947 saw five additions to the staff in Plant Pathology. Early in the year Roderick Sprague was added as Associate Professor and Associate Plant Pathologist in the Experiment Station and R. C. Lindner was appointed as Associate Plant Pathologist at the Tree Fruit Experiment Station to work on the physiology and biochemistry of stone fruit viruses. C. M. Wright was appointed as Assistant Plant Pathologist, with the State Department of Agriculture. Charles Gardner Shaw was appointed as Instructor and Junior Plant Pathologist. A. E. Rich was added to the staff as Instructor in Plant Pathology and Assistant Plant Pathologist to replace Stevenson who resigned to accept a position with the California Polytechnic Institute.

In 1948, S. O. Graham was appointed by the State Department of Agriculture to assist in the nursery improvement program, with Blodgett. Nyland resigned to accept appointment with the University of California at Davis. C. M. Wright resigned his position with the State Department of Agriculture to succeed Nyland. H. C. Kirkpatrick was appointed by the U. S. Department of Agriculture to work at the Tree Fruit Experiment Station in the field of chemotherapy of stone fruit viruses, with Lindner.

The year 1950 saw four staff changes. Sprague transferred from Pullman to the Tree Fruit Experiment Station at Wenatchee to conduct work on powdery mildew of apples and other parasitic types of tree-fruit diseases and also to be closer to the research on snow mold and snow scald of wheat in adjacent Douglas County. J. P. Meiners was appointed at Assistant Professor and Assistant Plant Pathologist to succeed Dr. Sprague. In the same year T. O. Diener was appointed as Assistant Plant Pathologist and stationed at the Irrigation Experiment Station at Prosser to work in the field of stone-fruit viruses, and J. G. Barrat was stationed at the same place as Plant Pathologist and Entomologist, succeeding S. O. Graham who resigned to continue graduate work.

At the present time there are 26 full-time plant pathologists in the State of Washington. Ten of these are at Pullman and the remainder at the various field stations throughout the State. Seven of the plant pathologists at Pullman are on the teaching staff and most of those at the outlying stations are listed with the Graduate Faculty to give recognition to their services in the counselling and instruction of graduate students. At the present time there are twelve graduate students in the Department, ten of whom are candidates for the doctor's degree.

Alumni

The Department of Plant Pathology has graduated 46 students with the Bachelor's Degree from the period 1912 through 1950. Sixteen have been awarded the degree Master of Science from 1916 through 1941. Seventeen have been awarded the Doctorate during the period 1930 through 1950. The names of these alumni, the degrees received and the year in which they were awarded are appended below.

Bachelor of Science

D. C. George	(In Bot.)	1912	Roderick Sprague	1924
B. F. Dana	(In Bot.)	1916	George A. Newton	1925
Walter J. Bach		1921	G. A. Huber	1925
L. W. Boyle		1923	E. G. Davenport	1926
D. J. Crowley		1923	John Large	1926
Charles S. Parker		1923	Harry Elcock	1926
Geo. D. Ruehle		1923	Jess Kienholz	1928
Chas. F. Lackey		1924	K. F. Baker	1930
C. H. Spiegelberg		1924	Lindsay Loring	1931

Bachelor of Science (Cont.)

Earl J. Anderson		1932	William E. Rader	1940
Henry Carl Newbom		1933	Elwood Fryer	1940
Carl Robert Frees		1933	George Nyland	1940
Otto F. Schnellhardt		1933	Edwin A. Bornander	1941
John A. Milbrath		1934	Ruth Halasey	1941
W. Harley English		1935	Hugh C. Kirkpatrick	1941
Richard H. Wellman		1935	John F. Schafer	1942
Karl E. Baur		1936	Neil A. MacLean	1943
Heston O. Weyrich		1936	Kirk Athow	1946
D. M. Coe		1937	Clarence H. Sonderman Jr.	1948
Carl K. Riesenweber		1938	Paul A. Johnson	1950
S. I. Cohen		1939	Allan R. Hoagland	1950
John R. Hardison		1939	Clarence Piper	1950
			John Y. Woo	1950

Master of Science

D. C. George	(In Bot.)	1916	Grover Burnett	1929
B. F. Dana	(In Bot.)	1917	G. A. Huber	1929
Walter J. Bach		1922	Jess R. Kienholz	1929
Chas S. Parker		1923	Kenneth J. Kadow	1933
L. W. Boyle		1924	Earl J. Anderson	1934
Carl H. Spiegleberg		1925	Otto F. Schnellhardt	1935
Roderick Sprague		1925	E. L. Reeves	1937
Geo. A. Newton		1927	Carl W. Boothroyd	1941

Ph.D.

George D. Ruehle	1930	S. I. Cohen	1943
G. A. Huber	1931	J. D. Menzies	1943
Grover Burnett	1932	Max L. Schuster	1945
Kenneth F. Baker	1934	George Nyland	1948
Leo Campbell	1935	Conley V. Lowther	1948
Folke Johnson	1939	Jack P. Meiners	1949
Richard H. Wellman	1939	Neil A. MacLean	1949
W. Harley English	1940	Avery E. Rich	1950
D. M. Coe	1943		

Present Teaching Program

At the present writing, sixteen courses of instruction are offered:

P. P. 9 Practical Plant Pathology. 3 hours.
P. P. 27 Forest Pathology. 4 hours.
P. P. 29 General Plant Pathology. 4 hours.
P. P. 115 Vegetable Diseases. 3 hours.
P. P. 117 Diseases of Ornamental Plants. 3 hours.
P. P. 119 Fruit Diseases. 3 hours.
P. P. 121 Diseases of Field Crops. 4 hours.
P. P. 128 Mycology. 4 hours.
P. P. 129 Methods and Techniques. 4 hours.
P. P. 150 Field Mycology. (summer course) 2-3 hours.
P. P. 215 Seminar. 1 hour.
P. P. 219 Advanced Principles and Theory of Plant Pathology.
P. P. 228 Advanced Mycology.
P. P. 250 Field Plant Pathology (summer course) 2-3 hours.
P. P. 300 Research
P. P. 350 Thesis

Front row, left to right:
H. B. Busdicker,
C. J. Gould,
G. W. Fischer,
R. C. Lindner,
D. M. McLean,
Folke Johnson.
Second row, left to right:
M. R. Harris,
W. D. Courtney,
S. B. Locke,
Roderick Sprague,
Leo Campbell,
A. E. Rich,
J. D. Menzies.
Third row, left to right:
W. D. Yerkes,
J. P. Meiners,
R. C. Cummings,
S. W. Graham,
H. E. Williams,
E. C. Blodgett,
T. O. Diener,
J. G. Barrat.
Fourth row:
C. M. Wright,
H. H. V. Hord.
Absent:
C. S. Holton,
E. L. Reeves,
H. C. Kirkpatrick,
T. R. Wright.

FIGURE 1. Washington State Plant Pathologists. Picture taken on occasion of Washington Agricultural Experiment Station Conference, November 27, 1950.

Present Personnel

Although the formal teaching program is confined to the campus at Pullman, the research program is rather widely decentralized. Of the 26 full-time plant Pathologists in the State of Washington, ten are on the Pullman campus, five are at the Tree Fruit Experiment Station at Wenatchee, five are at the Irrigation Experiment Station at Prosser, four at the Western Washington Experiment Station at Puyallup, and one each at the Northwest Washington Experiment Station at Mount Vernon and the Cranberry-Blueberry Experiment Station at Long Beach. The complete roster of plant pathologists follows:

List of Full-time Plant Pathologists in the State of Washington
(As prepared for Office of Experiment Stations)

G. W. Fischer, Professor; Plant Pathologist; Chairman of Department; Agricultural Experiment Station, Pullman.
J. G. Barrat, Plant Pathologist and Entomologist, Washington State Department of Agriculture, Irrigation Experimental Station, Prosser.
E. C. Blodgett, Plant Pathologist, Irrigation Experiment Station and Washington State Department of Agriculture, Prosser.
H. B. Busdicker, Pathologist, U. S. Department of Agriculture, Bureau of Entomology and Plant Quarantine, Pullman.
Leo Campbell, Assistant Plant Pathologist, Western Washington Experiment Station, Puyallup.
Wm. Bridge Cooke, Research Associate in Plant Pathology (Mycology), Pullman.
W. D. Courtney, Associate Nematologist, U. S. Department of Agriculture, Bureau of Plant Industry, Soils, and Agricultural Engineering, Western Washington Experiment Station, Puyallup.
D. J. Crowley, Associate Plant Pathologist, Cranberry-Blueberry Experiment Station, Long Beach.
T. O. Diener, Assistant Plant Pathologist, Irrigation Experiment Station, Prosser.
C. J. Gould, Associate Plant Pathologist, Western Washington Experiment Station, Puyallup.
M. R. Harris, Extension Plant Pathologist, Agricultural Extension Service, Pullman.
C. S. Holton, Pathologist, U. S. Department of Agriculture, Agricultural Experiment Station, Pullman.
Folke Johnson, Associate Plant Pathologist, Western Washington Experiment Station, Puyallup.
H. C. Kirkpatrick, Associate Pathologist, U. S. Department of Agriculture, Bureau of Plant Industry, Soils and Agricultural Engineering, Tree Fruit Experiment Station, Wenatchee.
R. C. Lindner, Associate Plant Pathologist, Tree Fruit Experiment Station, Wenatchee.
S. B. Locke, Associate Professor and Associate Plant Pathologist, Agricultural Experiment Station, Pullman.
D. M. McLean, Associate Pathologist, U. S. Department of Agriculture, Bureau of Plant Industry, Soils, and Agricultural Engineering, Northwest Washington Experiment Station, Mount Vernon.
J. P. Meiners, Assistant Professor and Assistant Plant Pathologist, Agricultural Experiment Station, Pullman.
J. D. Menzies, Pathologist, U. S. Department of Agriculture, Irrigation Experiment Station, Prosser.
E. L. Reeves, Pathologist, U. S. Department of Agriculture, Box 99, Wenatchee,
A. E. Rich, Assistant Professor and Assistant Plant Pathologist, Agricultural Experiment Station, Pullman.
C. G. Shaw, Assistant Professor, and Assistant Plant Pathologist, Agricultural Experiment Station, Pullman.
Roderick Sprague, Plant Pathologist, Tree Fruit Experiment Station, Wenatchee.
H. E. Williams, Junior Plant Pathologist, Irrigation Experiment Station, and Washington State Department of Agriculture, Prosser.
C. M. Wright, Assistant Professor and Assistant Plant Pathologist, Agricultural Experiment Station, Pullman.
T. R. Wright, Associate Pathologist, U. S. Department of Agriculture, Box 99, Wenatchee.

Present Research Program

The research program itself is divided on the basis of various crops, insofar as this is possible. The distribution of responsibility indicated below includes service responsibilities as well as research in the various categories.

CEREAL DISEASES

Wheat:

Bunt, including "stunt bunt or short smut" -- C. S. Holton (Physiologic specialization, inheritance of pathogenicity and comparative studies with similar grass smuts)

Flag smut -- C. S. Holton (General physiological studies)

Snow mold and snow scald -- Roderick Sprague (General biology and epidemiology of the causal organisms; control)

Root rots -- J. P. Meiners (General biological factors influencing the incidence of these diseases)

Stem rust -- H. B. Busdicker (Barberry eradication)

Barley:

Covered smut -- C. S. Holton (General biology)

Oats:

Loose and covered smuts -- C. S. Holton (Physiologic specialization; general biology of the causal organisms

Corn:

Head smut -- G. W. Fischer and C. J. Mankin (Biology and epidemiology)

POTATO DISEASES

Leaf roll -- S. B. Locke (Varietal resistance and susceptibility in cooperation with U. S. Department of Agriculture and the local Department of Horticulture; chemotheraphy; nature of leafroll resistance; varietal resistance to net necrosis development). Avery E. Rich (Field and storage factors influencing net necrosis; field factors influencing the incidence of leafroll). J. D. Menzies (Varietal resistance and susceptibility; comparative studies with other virus diseases confused with leaf roll)

Scab -- J. D. Menzies (Influence of soil treatments and rotation on the incidence of scab; distribution of the organism in virgin soils). A. E. Rich (Influence of soil treatments on the incidence of the organism and severity of the disease, cooperative with J. D. Menzies)

Miscellaneous virus diseases -- J. D. Menzies, A. E. Rich

Late blight and early blight -- Avery E. Rich (Fungicidal control; field factors influencing incidence)

Seed-piece decay -- Avery E. Rich

Storage decays and tuber defects -- Avery E. Rich and E. C. Blodgett

DISEASES OF OTHER VEGETABLES

Seed crops -- cabbage and beets -- D. M. McLean (White blight (Sclerotinia) of cabbage; mosaic of beets)

Spinach -- C. M. Wright and W. D. Yerkes (Downy mildew in the Walla Walla district)

Beans -- Leo Campbell (Rust; Botrytis blight; white blight)

Beets -- Leo Campbell (Black root)

Squash decays (in storage) -- Leo Campbell

Seed treatment -- C. J. Gould (Under Western Washington conditions)

DISEASES OF OTHER FIELD CROPS

Beans and peas -- J. P. Meiners (Blights and root rots)

Alfalfa and sweetclover -- J. P. Meiners

DISEASES OF STONE FRUITS

Virus diseases

Nursery stock improvement -- E. C. Blodgett, H. E. Williams, T. O. Diener, J. G. Barrat

Indexing studies -- E. C. Blogdett, H. E. Williams, T. O. Diener, H. C. Kirkpatrick

Transmission studies -- E. L. Reeves (Graft and bud transmission) E. C. Blodgett, H. C. Kirkpatrick, R. C. Lindner
General identification studies of stone fruit viruses -- E. L. Reeves, E. C. Blodgett, Folke Johnson, T. O. Diener
Physical and chemical identification of stone fruit viruses -- R. C. Lindner (Ultra-violet absorption spectra; Polyphenol tests)
Chemotheraphy (H. C. Kirkpatrick, R. C. Lindner)
Physical treatments for control of viruses -- H. C. Kirkpatrick, R. C. Lindner, E. C. Blodgett, H. E. Williams
Purification studies -- R. C. Lindner (Use of biochemical tests for the separation of individual components of a complex of viruses whose combined action gives certain definite recognized disease symptoms)
Effect of temperature on symptom expression -- H. C. Kirkpatrick (Determination of optimum temperatures for expression of disease symptoms)
Identification of viruses transmitted by insects -- R. C. Lindner (In cooperation with entomologists in their insect vector studies)
In vitro culture of viruses -- R. C. Lindner
Relationship of understocks to the expression of virus diseases -- E. C. Blodgett, T. O. Diener, J. G. Barrat, H. E. Williams

Brown rot -- Folke Johnson (Fungicidal control)
Peach leaf curl -- Folke Johnson (Fungicidal control)

DISEASES OF POME FRUITS

Apple mildew -- Roderick Sprague (Fungicidal control and life history studies)
Other fungus diseases of fruit trees -- Roderick Sprague
Fire blight -- Roderick Sprague
Decays in storage and transit -- T. R. Wright

DISEASES OF SMALL FRUITS

Strawberries -- Leo Campbell and Folke Johnson (strawberry yellows, "dud", and control of yellows and other virus diseases through suppression of insect vectors in cooperation with local entomologists)
Cane fruits -- Folke Johnson (rusts, blights, leaf spots, etc.)
Cranberries and blueberries -- J. D. Crowley, Folke Johnson (Fungicidal control of fungus diseases; influence of harvesting methods on the incidence of storage decays in cooperation with the Department of Agricultural Engineering)

DISEASES OF HAY AND PASTURE GRASSES

Turf diseases -- J. P. Meiners
Smuts and rusts -- G. W. Fischer, J. P. Meiners, S. M. Dietz, and W. N. Siang
Leaf spots -- Roderick Sprague, J. Y. Park

DISEASES OF ORNAMENTALS

Bulbous ornamentals -- C. J. Gould (in western Washington)
Greenhouse ornamentals -- C. M. Wright (chiefly in eastern Washington)

DISEASES OF FOREST AND SHADE TREES

Ponderosa pine blight -- Charles Gardner Shaw, George W. Fischer (caused by industrial gases)
Tar spot of large leaf maple -- George W. Fischer, John Y. Woo
Wood decay fungi -- C. G. Shaw, Wm. Bridge Cooke, H. H. V. Hord
Miscellaneous -- Charles Gardner Shaw

MISCELLANEOUS

Soil microflora of Columbia Basin virgin soil in relation to plant disease incidence -- J. D. Menzies
Peppermint rust -- Leo Campbell (Epidemiology and control; hife history studies)
Mycology -- Charles Gardner Shaw, Wm. Bridge Cooke
Enzyme chemistry of certain wood decays -- Charles Gardner Shaw, H. H. V. Hord
Bio-assay of ergot alkaloids produced in artifical culture -- C. M. Wright, R. H. Cummings (in cooperation with University of Washington)

Physiology of plant gall development -- S. B. Locke, Lafayette Frederick
Diseases caused by nematodes -- W. D. Courtney
Extension plant pathology -- M. R. Harris
Plant Introduction and Quarantine -- E. C. Blodgett, H. E. Williams
Influence of sprinkler irrigation on plant disease-- J. D. Menzies, J. G. Barrat
Soil-borne diseases as influenced by land management practices -- J. D. Menzies

SURVEY OF RESEARCH ACCOMPLISHMENTS

It is difficult to evaluate our own accomplishments subjectively, and believing that current work is better understood and will speak for itself, attention is given primarily in this section to achievements that preceded our present program.

We have, in the State of Washington, a rather closely knit and highly cooperative research program, involving participation by plant pathologists employed by Washington State College Experiment Stations, the United States Department of Agriculture, and the Washington State Department of Agriculture. When speaking, therefore, of the accomplishments of the Department, the participation of all three of these agencies is understood.

The written contributions to date number well over 500 scientific papers and bulletins, and more or less popular papers. In addition five books have been written by four members of the Department.

Outstanding among the research accomplishments is, of course, the contribution to the control of stinking smut or bunt of wheat. In the very early days of the Experiment Station, Professor Piper recorded the extreme prevalence and importance of wheat smut as causing tremendous losses to the wheat farmers. Furthermore, every year many threshing machines were ruined by explosion due to spontaneous ignition of the smut-laden atmosphere within the separator. The copper sulfate method of controlling wheat smut was proving ineffective and the formaldehyde treatment developed a few years later was not a great deal better. Dr. Heald and his associates demonstrated that this failure of seed treatment was due largely to "smut showers" resulting from the expulsion into the atmosphere of inestimable quantities of smut spores, which blew to nearby fallow fields and contaminated the soil. Special attention was given to methods of overcoming this soil contamination by cultural practices and date of seeding. Also, in cooperation with the local wheat breeders, much has been done toward the control of wheat smut through the breeding of resistant varieties. A number of years ago Robert Insinger, a prominent Spokane citizen, made the public statement that "If the State College of Washington had never done anything else except what they had contributed to the knowledge of methods of control of wheat smut, every cent which had been spent to date in the support of the College would have been justified." Numerous biological contributions have likewise been made in the study of the stinking smut of wheat. The work of Heald, Flor and Holton in demonstrating the existence of numerous physiologic races of wheat smut is now considered classic.

It is believed that the State of Washington can take credit for much of the knowledge on the nature and control of the Rhizoctonia disease of potatoes. Likewise, many studies have been made on the complex nature of the potato viruses. A classic contribution is the demonstration of the combined action of the vein-banding virus of potatoes and the latent virus to produce the destructive virus disease known as rugose mosaic. It is believed, also, that more recent contributions on the nature of potato leafroll and factors influencing its development, including net necrosis, are outstanding.

Much attention was given in the earlier years to the disease then known as western blight of tomato. Fifty to 75 percent losses due to this disease were common. For many years it was believed that the disease was caused by a fungus or bacterial organism, and species of Fusarium and Rhizoctonia were consistently isolated from the roots of the blighted tomato plants. At one time, it was published that the western blight was due to Fusarium oxysporum. Further work failed to confirm this, however, and it was then decided that a species of Rhizoctonia was the causal organism. Following Dr. Heald's arrival further investigations indicated that the disease was of virus nature, and it was later shown that the western blight of tomato is actually caused by the common curly top virus.

The streak disease of greenhouse tomatoes had long baffled growers and plant pathologists alike. L. K. Jones showed that this disease was due to tobacco mosaic transmitted to the tomato plants by greenhouse workers who used tobacco in one form or another.

Numerous contributions have been made by Heald and graduate students on the etiology and control of storage decays of Washington apples and pears. The most destructive of these were rots caused by blue mold and gray mold.

Also in the earlier years, many contributions were made on the identity, nature, and control of oat smuts in Washington, primarily by Heald and his associates.

Dr. Heald's demonstration of leaf invasion by fire blight bacteria is considered classic.

Leaf curl and mosaic, two virus diseases of geranium, had become prevalent in commercial stock of this greenhouse ornamental. Studies on these diseases showed that losses may be greatly reduced through careful selection of disease-free stock for propagation. Likewise, streak and mosaic, two virus diseases of cineraria, were studied in the earlier years. Jones showed that both of these diseases are seed-carried, and that the diseases can be controlled by careful selection of seeds from disease-free plants.

The nature and mode of transmission of the virus disease, witches' broom of alfalfa, was worked out by Menzies in association with Heald.

Campbell's study of black root of beets has shown that the disease can be controlled by crop rotation.

A great contribution was made toward the solution of the life history of the organism causing witches' broom of service berry. This was accomplished by Sprague in collaboration with Heald.

Several contributions have been made toward the solution of several disease problems affecting field peas and also green canning peas. Prominent among these are the Fusarium wilt, mosaics, and downy mildew, by Jones, Johnson and Campbell.

Another classic contribution was the demonstration by Heald and Sprague of the relationship between the incidence of silver leaf of apple trees (caused by Stereum purpureum) and winter injury.

Through the earlier work of Jones, Reeves, Menzies, and Huber, great strides were made toward a knowledge of the complex of virus diseases attacking our stone fruits and in methods of controlling them. These earlier contributions have been considerably augmented by recent work of Blodgett, Lindner, Kirkpatrick, Reeves, and their associates.

Many contributions have been made toward an understanding of various diseases of forage and range grasses by Sprague and Fischer.

The control of the destructive beet mosaic in northwest Washington seed beets has been accomplished by the simple method of isolating the foundation seed beet fields, based on the work of Pound and McLean. A similar contribution has been made toward the control of potato leafroll through the isolation of foundation fields for seed potato production, based on experiments by Rich and Locke.

Through the aggressive research activity of Gould many contributions have been made toward an understanding of the cause and control of various bulb diseases in western Washington.

The eradication of more than 120,000 barberry bushes from eastern Washington through the leadership of Busdicker is a noteworthy contribution by itself to the control of stem rust of wheat and other cereals.

Distinct contributions were made by Jones and later by Johnson on various diseases of raspberries, leaf rust (Phragmidium) of raspberries, and other bramble diseases.

Honorable mention is merited also by the work of the Moxee Plant Introduction and Quarantine Station under the earlier leadership of Coe, Menzies, and now Blodgett, to control virus diseases of stone fruit nursery stock by establishing and propagating virus-free bud wood sources.

Current contributions by the present staff are considered to be just as noteworthy as those of past years. However, we modestly refrain from "patting ourselves on the back". Some reference to current contributions will be made under the heading pertaining to the future.

? THE FUTURE ?

In these uncertain and troubled times any predictions for the future must be made with considerable reservations. In the foregoing pages we have seen the development of plant pathology in the State of Washington from the modest beginning where C. V. Piper was expected to handle the entire fields of Botany and Entomology in the College and Experiment Station in 1893, to the present time where we now have 26 full-time plant pathologists in the State with a great degree of subject-matter specialization. How much further development and specialization is possible and likely to occur is not safe for anyone to predict. In spite of the present comparatively large staff, there still remains a multiplicity of plant disease problems and all of the staff are probably as busy as were their much less specialized predecessors.

However, barring economic catastrophe, it would seem safe to predict that Lindner, Blodgett, Reeves, Kirkpatrick and their associates will continue to make outstanding contributions toward our knowledge of the components of the complex of virus diseases attacking stone

fruits. It can be predicted that Locke, Rich, and Menzies in cooperation with entomologists and horticulturists will continue to make contributions toward the identity and control of the components of the potato virus complex. Holton and his associates can be expected to solve the problem on dwarf smut of wheat, which now defies control except through the medium of resistant varieties. Campbell and cooperating entomologists will make further contributions on the control of strawberry yellows by suppressing the aphid vector. The current contributions toward the control of diseases of bulbous crops by Gould can be expected to continue.

It can also be expected that through the efforts of Menzies, McLean, and others much information will be obtained toward our knowledge of soil borne organisms and their relation to plant diseases and their control.

Present research on the effects of industrial gases on economic plants, by Shaw, Johnson, and cooperating chemists, can be expected to yield outstanding contributions in this currently important field.

Current research by Lindner, Kirkpatrick, and Locke on the chemotherapy of plant viruses, and by Lindner on chemical detection of viruses in plant tissues, has tremendous potentialities.

However, it must remain for the hindsight of someone in the future rather than the foresight of us in the present to record the present and future contributions of Washington State Plant Pathologists effectively.

DEPARTMENT OF PLANT PATHOLOGY, STATE COLLEGE OF WASHINGTON, PULLMAN

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 201

A KEY TO SPECIES OF HELMINTHOSPORIUM REPORTED ON GRASSES IN THE UNITED STATES

Supplement 201 May 15, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

A KEY TO SPECIES OF HELMINTHOSPORIUM REPORTED ON GRASSES IN THE UNITED STATES

E. S. Luttrell

Plant Disease Reporter
Supplement 201 May 15, 1951

A KEY TO SPECIES OF HELMINTHOSPORIUM REPORTED ON GRASSES IN THE UNITED STATES[1]

E. S. Luttrell

The purpose of the following key is to provide an index to the species of Helminthosporium found on grasses in the United States. Descriptions and a bibliography of the literature dealing with these species are available in Sprague's (1)[2] recently published book on diseases of cereals and grasses. Although at present a host index may afford the simplest means of identifying Helminthosporium species, a key based on the morphology of the fungus should be of practical value, especially in the determination of species on previously unreported hosts. This key is derived primarily from Drechsler's (2) detailed descriptions of the majority of the species with occasional reference to the more recent literature for emendations and for data concerning species reported since the publication of Drechsler's paper. It is based almost entirely on characteristics of the conidium since these characters may be utilized in the determination of material growing in culture as well as on the natural substrate. An attempt has been made to place primary emphasis on criteria which appear to be of major taxonomic importance. Consequently, the key may also serve in bringing together morphologically similar species and in emphasizing the relationships among them. The principal criteria employed are type of germination, shape, insertion of the hilum, wall thickness, color, diameter, length, and septation of the conidia. Often variations, especially in dimensions and color of the conidia, greater or lesser than those reported in the descriptions of species are found in individual collections, and the occurrence of morphologically distinct forms has been reported in several species. Consequently, difficulties in following the key are inevitable. These presently unavoidable imperfections in the key, however, should call attention to the type of data necessary for a satisfactory treatment of this group of fungi.

KEY TO SPECIES (3)

A. Conidia borne on sterigmata; perfect stage, when known, in Leptosphaeria.
The H. sigmoideum Group (4)

B. Producing large, spherical, black sclerotia 150-500 μ diam; conidia 29-49 x 10-14 μ, fusiform, curved, pointed at the ends, typically 3-septate, often constricted at the middle septum, intercalary cells brown, terminal cells greenish; causing leaf spot and culm rot of rice (Oryza sativa L.), also on Echinochloa and Zizaniopsis.
H. sigmoideum Cav.

B. Sclerotia irregular in shape, rough, smaller, 90-119 x 268-342 μ; conidia 41-58 x 9-12 u; less actively parasitic, on rice.
H. sigmoideum var. irregulare
Cralley & Tullis

A. Conidia sessile.

C. Conidia germinating from any or all cells, cylindric or subcylindric and tapering slightly toward the ends; perfect stage, when known, in Pyrenophora (= Pleospora).
The H. gramineum Group (Subgenus Cylindro-Helminthosporium of Nisikado, Genus Drechslera of Ito)

D. Conidia extremely large, typically over 300 μ long, 2-5 septate, in germination producing whorls of 3-4 thick germ tubes from slightly above the tip of the basal cell and slightly below the tip of the apical cell, occasionally producing germ tubes from intercalary cells, cylindrical, thin-walled, pale brown, 185-385 x 15-25 μ; causing zonate eye spot of Bermuda grass (Cynodon dactylon (L.) Pers.) and numerous other grasses.
H. giganteum Heald & Wolf (5)

---

[1]Paper No. 210, Journal Series, Georgia Experiment Station.
[2]Numbers in parenthesis refer to Notes and References, page 65.

D. Conidia typically less than 300 μ long, 1-7-11-septate, not producing germ tubes in whorls.

E. Conidia dark olivaceous, thick-walled, cylindric or subcylindric and tapering slightly toward both ends, 25-140 x 11-25 μ (av. 83 x 19 μ), 1-12-septate (av. 6.3); causing leaf spot of bluegrass (Poa pratensis L.) and other species of Poa.

H. vagans Drechsl.

E. Conidia subhyaline to pale brown or pale brownish- or yellowish-olivaceous, thin-walled (wall moderately thick in H. siccans).

F. Basal cell of conidium tapering, rounded cone-shaped.

G. Conidia subhyaline, 45-175 x 12-21 μ, 1-9-septate; causing leaf blight of Agropyron, wheat (Triticum aestivum L.), rye (Secale cereale L.) and other grasses.

H. tritici-repentis Died.

G. Conidia yellowish-brown to pale yellowish-brown, 28-183 x 9-22 μ, 0-10-septate; causing yellow spot of leaves of wheat.

H. tritici-vulgaris Nisikado

F. Basal cell of conidium rounded, hemispherical to hemi-ellipsoidal.

H. Conidia usually subcylindric and tapering decidedly toward the ends, less commonly cylindric.

I. Subcylindric conidia often widest near the apex and tapering toward the base although commonly obclavate, wall moderately thick, subhyaline to yellowish- or brownish-olivaceous, 31-131 (-151) x 11-20 μ (av. 80 x 17 μ), 2-10-septate (av. 4.9); causing leaf blight of ryegrass (Lolium spp.)

H. siccans Drechsl. (6)

I. Subcylindric conidia consistently obclavate, widest near the base and tapering toward the apex, wall thin.

J. Conidia often terminating in a constricted, sparingly septate apical prolongation.

K. Apical prolongation often bearing a conidial scar indicating an Alternaria-like formation of secondary conidia; conidia 30-200 x 14-18 μ, 1-10-septate, subhyaline to pale yellowish; causing leaf blight of Cinna and Sudan grass (Sorghum vulgare var. sudanensis (Piper) Hitchc.)

H. catenarium Drechsl. (6)

K. Apical prolongation not bearing a conidial scar; conidia 53-135 x 15-23 μ, 1-11-septate, subhyaline to yellowish; on withered leaves of Agrostis and Eleusine.

H. stenacrum Drechsl. (6)

J. Conidia rounded at the apex, not terminating in a constricted prolongation, subhyaline to yellow or fuligenous, 21-121 (-161) x 11-20 μ (av. 75 x 16 μ), 1-11-septate (av. 4.4); causing netblotch of fescue (Festuca spp.).

H. dictyoides Drechsl. (6)

H. Conidia usually cylindric, less commonly subcylindric and tapering slightly toward the ends.

L. Maximum diameter of conidia 18-24 μ.

M. Conidia usually slightly constricted at the septa (especially the basal septum, this constriction producing a subglobose basal cell), subhyaline to greenish-fuligenous or yellow, 30-175 x 15-22 μ, 1-11-septate; causing netblotch of barley (Hordeum vulgare L.).
H. teres Sacc. (6)

M. Conidia usually not constricted at the septa.

N. Conidia of medium size, maximum length less than 200 μ.

O. Conidia 31-141 (-170) x 12-20 μ (av. 88 x 17 μ), 1-11-septate (av. 4.9), olivaceous to yellowish-brown; causing leaf blotch of oats (Avena sativa L.).
H. avenae Eidam (6)

O. Conidia 31-111 (-131) x 11-24 μ (av. 70 x 17 μ), 1-7-septate (av. 4.9), subhyaline to yellowish-brown; causing stripe of barley.
H. gramineum Rabh. (6)

N. Conidia large, maximum length over 200 μ (under moist conditions up to 400 μ), pale brown, 45-265 x 14-26 μ, 1-10-septate; causing leaf spot of brome grasses (Bromus spp.).
H. bromi Drechsl. (6)

L. Maximum diameter of conidia 16 μ; conidia yellowish to pale olivaceous, 21-105 x 8-16 μ (av. 65 x 12)μ, 2-10-septate (av. 5.4); causing leaf spot of Agrostis.
H. erythrospilum Drechsl. (6)

C. Conidia germinating by polar germ tubes, ellipsoid to fusoid; perfect stage, when known, in Cochliobolus (= Ophiobolus).
The H. sativum Group (Subgenus Euhelminthosporium of Nisikado).

P. Conidia with protruding conical hilum.

Q. Basal cell or basal and apical cells of conidia frequently lighter in color and set off by accentuated, thicker, darker septa.

R. Apex of conidium often produced as a long, constricted, cylindrical rostrate extension; conidia 32-184 x 14-22 μ, 8-15-septate, dark olivaceous; causing leaf spot of Eragrostis, Paspalum, sorghum (Sorghum vulgare Pers.), corn (Zea mays L.), Sudan grass, pearl millet (Pennisetum glaucum (L.) R.Br.)
H. rostratum Drechsl.

R. Apex of conidium not rostrate.

S. Conidia 20-105 x 10-14 μ, 1-12-septate, brownish-yellow to dark olivaceous; mold and secondary seed and root rot of Distichlis and other grasses.
H. halodes Drechsl.

S. Conidia 23-72 x 13-20 μ (av. 52 x 16.5 μ) 2-9-septate (av. 6), light greenish-brown to smoky-brown; possibly occurring on roots of various grasses in the United States.
H. halodes var. tritici Mitra.

Q. Basal and apical cells of conidia concolorous with intercalary cells, not set off by accentuated septa.

T. Maximum diameter of conidia less than 20 μ, maximum length less than 100 μ; conidia subcylindric-fusoid to ellipsoid, straight or slightly curved, subhyaline to pale fuligenous with hyaline cone-shaped area around hilum, 28-92 x 10-18 μ, 3-9-septate; causing leaf blight of Paspalum.

H. micropus Drechsl.

T. Maximum diameter of conidia over 20 μ, maximum length over 100 μ.

U. Conidial wall thin; conidia subhyaline to pale fuligenous, later moderately fuligenous, greenish-yellow, yellowish-brown, or pale olive, 45-132 x 15-25 μ, 1-8-septate, fusoid, straight or slightly curved; causing leaf blight of corn, sorghum, Johnson grass (Sorghum halepense (L.) Pers.), Sudan grass.

H. turcicum Pass.

U. Conidial wall thick; conidia dark olivaceous, yellowish when young, 40-150 x 15-22 μ, 3-10-septate, fusoid, straight or curved (curvature often pronounced); causing spot blotch of Echinochloa.

H. monoceras Drechsl.

P. Conidia without protruding hilum; hilum included within the contour of the wall of the basal cell.

V. Conidia 1-6-septate.

W. Conidia yellowish, ellipsoid, often broader near the apex and tapering to the pointed base, frequently constricted at the basal septum, 18-48 x 8.5-14 μ, 2-6-septate; on withered leaves of Agrostis and Anthoxanthum.

H. dematioideum Bub. & Wröbl.

W. Conidia dark olivaceous to brown.

X. Maximum length of conidia more than 50 μ, maximum width less than 20 μ.

Y. Conidia ellipsoid to fusoid-obclavate (widest near the base), 15-67 x 11-17 μ, 1-6-septate, wall thick, dark olivaceous; on withered leaves of Eleusine and Eragrostis.

H. leucostylum Drechsl.
(= ? H. hadrotrichoides Ell. & Ev.)

Y. Conidia fusoid-clavate (widest near the apex), 22-78 x 12-19 μ, 1-5-septate, brown; causing false smut of Sporobolus.

H. ravenelli Curt.

X. Maximum length of conidia less than 50 μ, maximum width 20 μ or more.

Z. Conidia 20-41 x 10-20 μ, mostly 3-septate, dark olivaceous to brown, ellipsoid; producing long, simple or branched, pseudoparenchymatous stromata in culture; causing root rot of wheat.

H. tetramera McKinney (7)

Z. Conidia 35-50 x 15-21 μ, 2-3-septate, dark olivaceous, ellipsoid, wall thick; not producing stromata; mold of Agrostis and Dactylis.

H. triseptatum Dreschs. (7)

V. Conidia 1-7-13-septate

(a) Conidia dark olivaceous, wall thick.

(b) Basal cell of conidium prolonged as a cylindrical, pedicel-like protuberance; conidia broad-fusoid, typically straight, brownish-loive, hyaline at tip of

basal cell in area surrounding hilum, 31-91 x 15-29 μ (av. 65 x 23 μ), 1-9 septate (av. 7); isolated from roots of wheat.

H. pedicellatum A.W. Henry (Minnesota Agr. Expt. Sta. Tech. Bull. 22: 42, 1924).

(b) Basal cell of conidium not protuberant.

(c) Conidia usually straight.

(d) Hilum extremely large, conspicuous; producing columnar pseudoparenchymatous stromata bearing conidiophores toward the apex in culture; conidia subcylindric to fusoid or ellipsoid, dark olivaceous except in restricted subhyaline areas at the tips, 45-110 x 12-17 μ, 4-12-septate; causing leaf spot of Danthonia.

H. cyclops Drechsl.

(d) Hilum moderately conspicuous, not producing columnar stromata in culture.

(e) Maximum diameter of conidia less than 20 μ.

(f) Maximum diameter of conidia 14 μ; conidiophores arising from a tuberous erumpent stroma; conidia oblong, rarely subclavate, olivaceous, straight, thick-walled, 50-100 x 12-14 μ, 4-7-septate; on rotted culms of rye.

H. tuberosum Atk.

(f) Maximum diameter of conidia 17-18 μ; conidiophores not arising from a tuberous stroma.

(g) Conidia 25-103 x 7-18 μ (av. 63 x 13 μ), 2-12-septate (av. 7), fusoid, straight, or slightly curved, dark olivaceous-brown; causing leaf spot and ear rot of corn.

H. carbonum Ullstrup (8)

(g) Conidia 33-115 x 10-17 μ, 3-11-septate-, fusoid, straight or slightly curved, olivaceous to black-olivaceous or chocolate brown; saprophytic on corn stalks.

H. zeicola Stout (8)

(e) Maximum diameter of conidia more than 20 μ; conidia 40-85 x 20-26 μ (av. 58 x 22 μ), 3-9-septate, fusiform, straight, dark olivaceous; causing rusty blotch of leaves of barley.

H. californicum Mackie & Paxton (8)

(c) Conidia usually curved.

(h) Maximum diameter of conidia 18 μ; conidia fusiform-obclavate to ellipsoid, usually curved, 40-120 x 10-18 μ, 5-10 septate, dark olivaceous, occasionally paler in the terminal cells, causing root rot and spot blotch of Setaria.

H. setariae Saw. (8)

(h) Maximum diameter of conidia 20 μ or more.

(i) Causing spot blotch and blight of cereals and grasses.

(j) Causing spot blotch and root rot of barley and numerous other grasses; conidia fusiform, typically curved, dark olivaceous; 60-120 x 15-20 μ, 3-10-septate.

H. sativum Pam., King, & Bakke (8)

(j) Causing blight of Victor Grain oats; conidia fusiform, slightly curved, fuligenous to dark olivaceous, 40-130 x 11-25 µ (av. 70 x 15 µ), 4-11-septate (av. 8).
H. victoriae Meehan & Murphy (8)

(i) Causing brown leaf stripe of sugarcane (Saccharum officinarum L.); conidia fusiform, typically curved, dark-olivaceous, 40-133 x 12-22 µ (av. 83 x 17 µ), 3-12-septate (av. 7.7).
H. stenospilum Drechsl. (8)

(a) Conidia fuligenous to brown, wall relatively thin.

(k) Conidia terminating in a long, slender, apical, flagellum-like process, ellipsoid, fuligenous, 70-150 µ long, 7-9-septate; on leaves of Panicum.
H. flagelloideum Atk.

(k) Conidia not terminating in a flagellum-like process.

(l) Maximum diameter of conidia less than 15 µ, maximum length less than 100 µ.

(m) Maximum diameter of conidia 11 µ; conidia obclavate, rounded at the base, tapering to the apex, straight or curved, fuligenous to dark fuligenous, 27-86 x 8-11 µ (av. 52 x 10 µ), 2-9-septate; causing leaf spot of buffalo grass (Buchloë dactyloides (Nutt.) Engelm.) and grama grasses (Bouteloua spp.)
H. buchloës Lefebvre & A.G. John.

(m) Maximum diameter of conidia 14 µ; conidia ellipsoid to fusoid.

(n) Germ tubes branching at some distance from the conidium; conidia 50-95 x 11-14 µ, 3-12-septate, ellipsoid, slightly curved, pale fuligenous; causing leaf spot of Leersia.
H. leersiae Atk.

(n) Germ tubes branching close to the conidium; conidia 27-80 x 11-14 µ, 3-9-septate, subcylindric-fusoid, straight or slightly curved; causing leaf spot of Bermuda grass, also on Eleusine and Muhlenbergia.
H. cynodontis Marig.

(l) Maximum diameter of conidia more than 17 µ, length more than 100 µ.

(o) Conidia up to 170 µ in length, fusiform, curved, fuligenous to brownish, 16-90 (-170) x 11-17 µ, 5-8-(13)-septate; causing leaf spot, culm and inflorescence damage, and seedling blight of rice.
H. oryzae V. Breda de Haan (9)

(o) Conidia not over 115 µ in length.

(p) Conidia yellow or pale olivaceous.

(q) Average number of conidial septa 7.7-8.3; conidia fusiform, curved, fuligenous to pale olivaceous, 25-127 x 7-21 µ, (av. 89 x 15 µ), 3-13-septate; causing leaf spot of corn.
H. maydis Nisik. & Miy. (9)

(q) Average number of conidial septa 5.1; conidia smaller, less strongly curved, fusiform, yellow to pale olivaceous, 20-105 x 8-20 µ (av. 59 x 14 µ), 2-8-septate; causing leaf spot of sorghum, Johnson grass, Sudan grass.
H. sorghicola Lefebvre & Sherwin (9)

(p) Conidia brown.

(r) Conidia 19-132 x 8-21 μ, 2-11-septate, fusiform, curved, pale to dark brown; causing eye spot of sugarcane, Napier grass (Pennisetum purpureum Schumach.), and lemon grass (Cymbopogon citratus (DC.) Stapf).
H. sacchari (V. Breda de Haan) Butler (9)

(r) Conidia 50-100 x 12-17 μ, 4-10-septate, fusiform, straight or curved and flattened on the inner side, brown; causing leaf spot and blight of Eleusine.
H. nodulosum (Berk. & Curt.) Sacc. (9)

Notes and References

1. Sprague, Roderick. Diseases of cereals and grasses in North America. New York. Ronald Press Co., 538 pp., 1950.

2. Drechsler, Charles. Some graminicolous species of Helminthosporium. Jour. Agr. Res. 24: 641-739. 1923.

3. According to the description by Saccardo (Syll. Fung. 4: 408, 1886), H. parvulum Cke., which was listed by Weiss (Index of Plant Diseases in the United States. U. S. D. A. Plant Dis. Survey Special Pub. No. 1, Part 3, p. 420, 1950), on pampas grass, is doubtfully a Helminthosporium species. Certainly the description, "conidia scattered, cylindrical, obtuse, 1-2-septate, straight, brown, 10-17 x 3.4 μ", is too incomplete to permit the inclusion of this species in the key. H. poae Baudýs, which was reported by Sprague (l.c. (1)) on species of Poa, likewise is inadequately described and is, therefore, omitted from the key. According to Saccardo (Syll. Fung. 25: 823, 1931) the conidia of H. poae are cylindrical, pale brown, 36-73 x 17-22 μ, and 2-6-septate with thick septa. Although H. flagelloideum and H. tuberosum apparently have not been reported in the United States since they were originally described by Atkinson (Bull. Cornell Univ. (Sci.) 3 (1), 50 pp., 1897) and are inadequately known, they are tentatively inserted in the key. The flagellum-like apical process of the conidium appears to be sufficient to distinguish H. flagelloideum from other species of Helminthosporium. However, the tuberous stromata which Atkinson described as the most important characteristic of H. tuberosum possibly represent immature perithecia on which conidiophores are produced, a type of development which occurs in H. teres also according to Drechsler (l.c. (2)). It is probable that the conidiophores of H. tuberosum normally arise directly from the leaf or stem surface; and, in this case, Atkinson's description offers little basis for distinguishing H. tuberosum from related species.

4. The single species and one variety in this group probably should be placed in a separate genus. H. sigmoideum is clearly distinct from other species of Helminthosporium on grasses in the production of sterigmata and falcate, acutely-pointed conidia.

5. Because of its peculiar type of germination and its production in culture of a Hormodendron-like stage from the hyphae or, by proliferation, from the conidia, Drechsler (Jour. Agr. Res. 37: 473-492, 1928) expressed doubt that H. giganteum should be included in the same series with other species placed in the Cylindro-Helminthosporium group. It is readily distinguishable from any other species of Helminthosporium on grasses.

6. H. siccans, H. catenarium, H. stenacrum, H. dictyoides, H. teres, H. avenae, H. gramineum, H. bromi, and H. erythrospilum represent a complex of similar species. H. vagans differs decidedly from these species in the thicker walls and deeper color of the conidia. H. tritici-repentis and H. tritici-vulgaris, two nearly identical species, are distinguished only by the peculiar shape of the basal cell of the conidium. It is uncertain whether the constricted apical prolongation, the character utilized in the separation of H. stenacrum and H. catenarium, is peculiar to these two species or whether it is an

aberration which may be induced in other species as well under certain environmental conditions. A proliferation to form secondary conidia similiar to that in H. catenarium has been reported in H. sorghicola by Lefebvre and Sherwin (Mycol. 40: 708-716, 1948) and in H. gramineum, H. teres, H. cyclops, and H. sativum by Drechsler (l.c. (2)). In spite of this peculiarity, therefore, H. stenacrum and H. catenarium appear to be closely related to the other species of the H. gramineum complex. Conidial shape appears to be a useful character in the separation of species in this complex, although differences can be expressed only as tendencies. In H. dictyoides expecially the consistently obclavate shape is a pronounced characteristic of the conidium. The moderately thick walls of fully mature conidia of H. siccans may assist in distinguishing this species. However, the walls often are not appreciably thicker than those of other species, and it is difficult to rely on this character. Constriction of the conidium, the character employed in the separation of H. teres, is of doubtful value since the constrictions in H. teres are slight and similiarly constricted conidia may occur in other species. Here again differences can be expressed only as tendencies. It may be necessary to depend principally on conidial dimensions and septation in the separation of species in this complex (Drechsler, Phytopath. 25: 344-361, 1935), but in view of the differences among races of H. gramineum reported by Christensen and Graham (Minnesota Agr. Expt. Sta. Tech. Bull. 95: 1-40, 1934) it is evident that more extensive data on variation within the species are necessary before such criteria can be used with confidence. The lesser diameter of the conidia of H. erythrospilum and the greater size of H. bromi conidia may be sufficient to distinguish these two species. It should be noted, however, that conidial dimensions of H. avenae as frequently reported (Sprague l.c. (1)) are similiar to those of H. erythrospilum, not exceeding 16 μ in diameter and hardly exceeding 100 μ in length. The larger dimensions for H. avenae given in the key are taken from Drechsler (Phytopath. 25: 344-361, 1935; l.c. (2)) who reported conidia of H. avenae as of approximately the same size as those of H. teres and clearly distinguishable by their greater diameter from those of H. erythrospilum.

7. H. tetramera and H. triseptatum are very close to the short-spored forms in the genus Brachysporium. If Brachysporium is to be maintained as a genus, it probably must be separated from Helminthosporium upon the basis of some arbitrarily selected conidial length/width ratio. Judging from the conidial dimensions of species included in Brachysporium by Saccardo (Syll. Fung. 4: 423-430, 1886) this ratio should be in the neighborhood of 3:1. On this basis H. tetramera and H. triseptatum, as well as some of the species placed in Helminthosporium by Saccardo himself, would fall in Brachysporium. However, it is doubtful that Brachysporium can be maintained as a separate genus. Drechsler (Jour. Agr. Res. 31: 701-726, 1925) suggested that many of the species in Brachysporium may be more closely related to species in the subgenus Euhelminthosporium than are the latter to species in the subgenus Cylindro-Helminthosporium.

8. H. carbonum, H. zeicola, H. californicum, H. setariae, H. sativum, H. victoriae, and H. stenospilum constitute a complex of closely related species, most of which are of doubtful validity. The separation of these species on the basis of curvature and diameter of the conidia is uncertain because of variations among different collections and strains within the species. The typically curved conidia of H. sativum, for example, may sometimes be predominantly straight; and in H. californicum, which Sprague (l.c. (1)) considered to be synonymous with H. sativum, they were described as being "not curved". Although H. carbonum is segregated with species in which the diameter of the conidia does not exceed 18 μ, Robles (Phytopath. 39: 1020-1028, 1949) reported the average dimensions of conidia of this species as 87 x 21 μ. Furthermore, he described, as "Helminthosporium Z", material from corn in which the conidia were 36-238 x 10-26 μ (av. 115 x 19 μ), 1-14-septate (av. 8), fusiform, usually slightly curved, and dark olivaceous brown. He believed that "Helminthosporium Z" might prove to be a form of H. carbonum. It seems probable that H. carbonum is a synonym of H. zeicola. The restricted parasitism of H. carbonum on certain recently developed inbred lines of corn might well indicate that it is a previously saprophytic fungus which adopted a parasitic habit when new hosts offering a more favorable substrate became available. It may be possible to distinguish H. setariae from H. sativum and H. stenospilum on the basis of its narrower conidia. However, Parris (Phytopath. 40: 90-103, 1950) reported the conidial dimensions of H. stenospilum as 52-133 x 10-16 μ (av. 85 x 14 μ), and these dimensions approximate those of H. setariae. H. victoriae apparently is only a physiologic race of H. sativum

and, along with H. californicum, probably should be reduced to synonomy with this species. This view is strengthened by Earhart's (Phytopath. 41: 11, 1951) report of a strain of Helminthosporium resembling H. sativum in some respects and H. victoriae in others and causing blight of an oat variety resistant to H. victoriae. Also, there is no evident morphological basis for the separation H. stenospilum from H. sativum.

9. H. oryzae, H. maydis, H. sorghicola, H. sacchari, and H. nodulosum are so closely related that they constitute hardly more than a group of physiologic forms of a single species. H. buchloës is distinguished from this group by the lesser dimensions and obclavate shape of its conidia. The nearly identical species H. cynodontis and H. leersiae are distinguished only by the smaller size of their conidia, and it should be noted that the conidial dimensions of some of the isolates of H. sacchari studied by Parris (Phytopath. 40: 90-103, 1950) fell within the range given for H. cynodontis. The difference in conidial length employed in the separation of H. oryzae is of doubtful value since, according to Drechsler (l.c. (2)), the larger conidia of this species are found only on the mats of hyphae covering the glumes of the host. A similiar development probably occurs in H. maydis. Drechsler (Jour. Agr. Res. 31: 701-726, 1925) described a fungus on moldy tassels of corn which he considered to be H. maydis, although the conidia were much longer than those of this species as they normally develop on leaves, measuring 28-155 x 10-17 μ. Differences in color as well as in extreme and mean dimensions and septation of the conidia in this group appear to be of questionable significance in view of the great variation reported by Parris (Phytopath. 32: 46-63, 1942; Ibid. 40: 90-103, 1950) in isolates of H. sacchari. He described forms in which the conidia varied from subhyaline through yellowish, fuligenous, or olive-green to dark brown, the latter approaching H. stenospilum in color. Drechsler (l.c. (2)) also reported the occurrence of dark olivaceous, thick-walled conidia in cultures of H. oryzae. Consequently, the distinction between the H. oryzae complex and the H. sativum complex is not as clear as might be desired. The criteria of color and especially wall thickness, however, are sufficient to distinguish typical forms of the two groups.

GEORGIA AGRICULTURAL EXPERIMENT STATION, EXPERIMENT, GEORGIA

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 202

SOME NEW AND IMPORTANT PLANT DISEASE OCCURRENCES AND DEVELOPMENTS IN THE UNITED STATES IN 1950

Supplement 202 May 15, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

SOME NEW AND IMPORTANT PLANT DISEASE OCCURRENCES AND DEVELOPMENTS IN THE UNITED STATES IN 1950

Compiled by Nellie W. Nance

Plant Disease Reporter
Supplement 202 May 15, 1951

This summary includes the important diseases of 1950 reported up to the present time, compiled for the most part from reports to the Plant Disease Survey and articles in Phytopathology.

The growing season over much of the country favored the development of many crop diseases. As a result of an extremely favorable cool-wet summer tomato late blight attained the most widespread distribution ever recorded in this country, with losses possibly exceeding those suffered from the destructive 1946 outbreak. The disease moved westward, appearing in States where it had not previously been reported. Heavy rains during the season resulted in crop damage caused by a number of diseases not usually of so much importance, e. g., Ascochyta blight of cotton in some Southern States, bacterial blights and anthracnose of beans in Michigan and others that will be noted.

On the other hand relatively light incidence of diseases on small grains in the Southeastern Coastal Plain was attributed to dry weather during the 1949-50 winter and spring season; in spite of the drought, all the major diseases were present in this area to a greater or less extent.

The weighted average temperature for the United States was 52.5° which is 0.7° below the 58-year mean. The precipitation averaged 29.68 inches, or 0.5 of an inch more than the 58-year mean of 29.18 inches. For detailed information on temperature and precipitation for the year see maps on page 71.

During 1950, through the cooperation of plant pathologists, the Plant Disease Survey started a series of Supplements to the Reporter (192, 195) on plant pathological investigations in the United States. The series was planned mainly to bring out the diversity of plant disease problems in this country and to show how the results of these investigations have affected plant disease occurrence and importance.

Papers presented at a symposium on teaching plant pathology during the Pittsburgh meetings of the American Phytopathological Society were published in the April issue of the Reporter. A second symposium on this topic was held at the New York meetings of the Society, and published in the September issue of the Reporter.

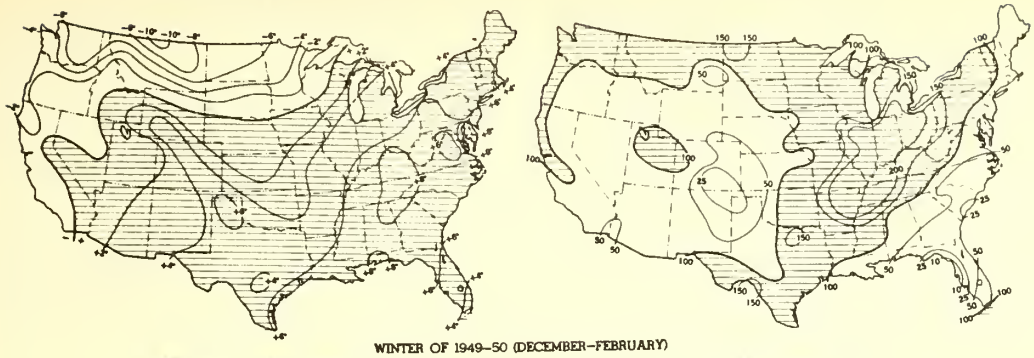
The 7th International Botanical Congress met at Stockholm, Sweden, July 6-20, 1950. The nomenclature section had before it for action 542 proposals involving changes in or additions to the International Rules of Botanical Nomenclature. Action taken at this Congress as it affects mycological nomenclature was published in the November issue of the Reporter.

Parts I, II, and III of the index of plant diseases in the United States compiled by Freeman Weiss were published in 1950 as Spec. Publ. Plant Dis. Surv. No. 1. Part I contains the list of plant diseases occurring in 43 families from Acanthaceae to Compositae, Part II deals with 31 families from Convolvulaceae to Gnetaceae, and Part III contains the Gramineae. The index is essentially an annotated host list, the members of each family being listed alphabetically under the Latin binomial, together with the common names and distribution. Under each is given a list of the diseases and their distribution.

TEMPERATURE AND PRECIPITATION

Departure of Mean Temperature from Normal

Percentage of Normal Precipitation



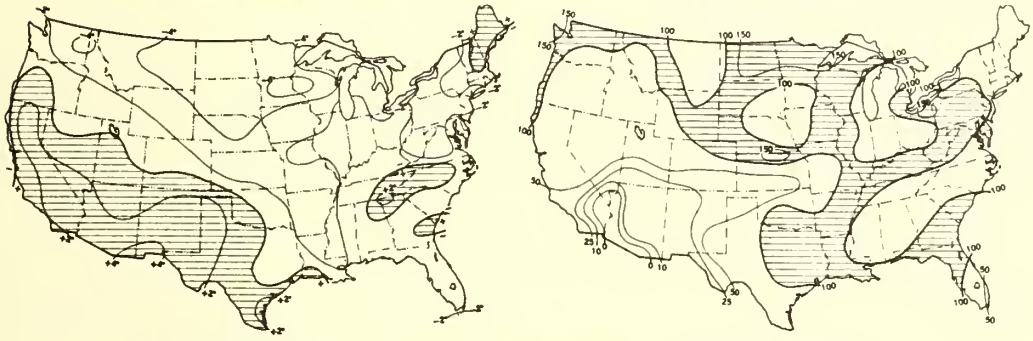
WINTER OF 1949–50 (DECEMBER–FEBRUARY)

Shaded Areas Normal or Above

Shaded Areas Normal or Above

Departure of Mean Temperature from Normal

Percentage of Normal Precipitation



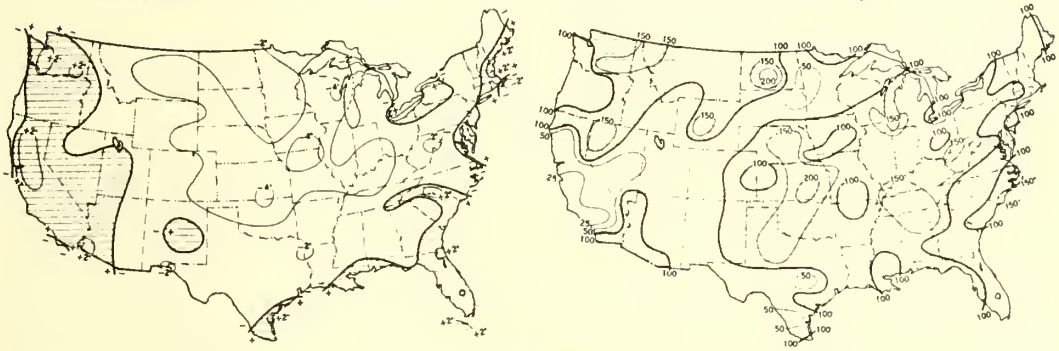
SPRING OF 1950 (MARCH–MAY)

Shaded Areas Normal or Above

Shaded Areas Normal or Above

Departure of Mean Temperature from Normal

Percentage of Normal Precipitation



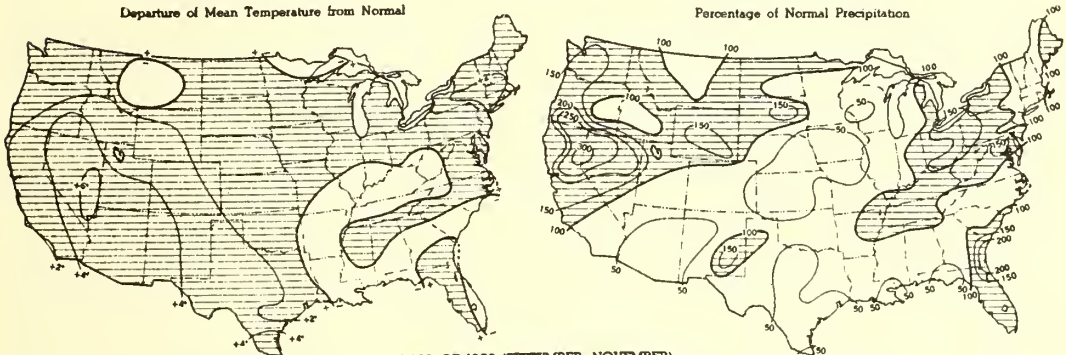
SUMMER OF 1950 (JUNE–AUGUST)

Shaded Areas Normal or Above

Shaded Areas Normal or Above

Departure of Mean Temperature from Normal

Percentage of Normal Precipitation



FALL OF 1950 (SEPTEMBER–NOVEMBER)

Shaded Areas Normal or Above

Shaded Areas Normal or Above

(From Weekly Weather and Crop Bulletin National Summary, Volume 37, 1950.)

During the year 26 diseases were reported in States where they had not been found before on a particular host (Table 1). Diseases found or reported in this country for the first time in 1950 or found on new hosts are listed in Table 2.

Table 1. Diseases reported in States where they had not been found or reported on a particular host until 1950.

Host Disease (Cause)	Where found	Remarks
BARLEY		
Net blotch (Pyrenophora teres)	New Mexico	Most of the plants in a five-acre field appeared diseased. (PDR 35: 168)
CORN		
Gray leaf spot (Cercospora zeae-maydis)	Virginia	(PDR 34: 394)
ALFALFA		
Alfalfa wilt (Corynebacterium insidiosum)	North Carolina	Endemic in one locality in the western mountain region of the State. (PDR 34: 205)
Stem nematode (Ditylenchus sp.)	North Carolina	Found in one alfalfa field in Harnett County, in 1949. (PDR 34: 205)
ALSIKE CLOVER		
Aster yellows ? (virus)	Oregon	Observed near Merrill during July 1950. Diseased plants formed no seeds, thus being a total loss. (PDR 34: 322)
LADINO CLOVER		
Leaf spot (Curvularia geniculata)	North Carolina	Found in several locations in 1949. (PDR 34: 205)
CITRUS SPP.		
Quick decline, tristeza (virus)	Louisiana	Found in an orange grove near Buras late in June 1950. (Florida Grower for Dec. 1950, p. 12)
Root nematode (Tylenchulus semipenetrans)	Texas	Nematodes found on roots of grapefruit tree near Mission. Six miles away the nematode was found on an orange tree. (PDR 34: 269)
PEACH		
Diplodia infection occurring naturally on peach fruit	Georgia	Found at Fort Valley, in 1950. (PDR 35: 221)
PERSIAN (ENGLISH) WALNUT		
Bacterial blight (Xanthomonas juglandis)	Illinois	Reported in Tazewell County. (PDR 34: 352)
AFRICAN VIOLET		
Powdery mildew (Oidium sp.)	Pennsylvania	Found on April 19, 1950 in Pittsburgh. (PDR 34: 197)
CAMELLIA		
Flower blight (Sclerotinia camelliae)	North Carolina	Fungus found at Winnabow. (PDR 34: 196)
	Louisiana	Found in Shreveport. (PDR 34: 188)

Table 1. (Continued)

Host Disease (Cause)	Where found	Remarks
ROSE Rust (Phragmidium americanum?)	Louisiana	Reported from Tensas Parish. First occurrence of rose rust in the Deep South. (PDR 34: 197)
SNAPDRAGON Powdery mildew (Oidium sp.)	Washington California	Caused economic damage to greenhouse-grown snapgragons. (PDR 34: 183)
TOBACCO Black shank (Phytophthora parasitica var. nicotianae)	Pennsylvania	Found on a farm in Lancaster County during summer of 1950. (PDR 35: 56)
Stem rot and barn rot (Pythium aphanidermatum)	South Carolina	Stem rot occurred on transplants. Barn rot most severe on bottom leaves harvested during wet weather. (Phytopath. 41: 17)
SUGAR BEET Sugar beet nematode (Heterodera schachtii)	Arizona	Generally distributed in one 34-acre field, in the Salt River Valley where about 1000 acres of sugar beets are grown annually for seed. (PDR 35: 173)
MAPLE Bleeding canker (Phytophthora cactorum)	Tennessee North Carolina	Reported on red maple in Cherokee National Forest in eastern Tenn. Found on one ornamental sugar maple in Morgantown, N. C. (PDR 35: 119)
AMERICAN ELM Dutch elm disease (Ceratostomella ulmi)	Illinois	The diseased elm was located in a roadside park on State Route 16. Specimens collected Aug. 3, 1950. (PDR 35: 56)
OAK Oak wilt (Chalara quercina)	Pennsylvania Kansas Illinois	The distribution of the disease in the State is not known. (PDR 34: 291) Reported on post oak in Douglas County, Kans. (PDR 35: 119) Found naturally infecting swamp white oak in Cook Co., Ill. First report of natural infection in woods. (PDR 35: 173)
SLASH PINE Cone rust (Cronartium strobilinum)	Georgia	Collected on cones of slash pine in vicinity of Pine Harbor during June of 1950. (PDR 34: 349)
SNOWBALL Spot anthracnose (Sphaceloma viburni)	Maryland	Infections found in Worcester and Somerset Counties. (PDR 34: 350)

Table 1. (Continued)

Host Disease (Cause)	Where found	Remarks
POTATO		
Pink rot (Phytophthora erythroseptica)	New York	Generally associated with excessively high moisture conditions. (PDR 35: 55)
Unmottled curly dwarf (virus)	New Hampshire	Reported on B 76-43 plants (U. S. D. A. variety), 1% showed symptoms. (PDR 34: 322)
SWEETPOTATO		
Internal cork (virus)	Oklahoma	Found in fall of 1949. In Jan. 1950 it was found in 11 out of 12 storage houses visited in widely scattered locations. (PDR 35: 227)
	California	Found in varietal plots at Davis and Shafter and in an experimental planting of Bunch Porto Rico at Manteca. (PDR 35: 230)
TOMATO		
Late blight (Phytophthora infestans)	Kansas	Found on fruits at Concordia, Cloud County, in Nov. 1950. (PDR 35: 120)

Table 2. Diseases found or reported in this country for the first time in 1950 = *; diseases found on new hosts = **.

Host Disease (Cause)	Where found	Remarks
BLUE LUPINE		
Powdery mildew ** (Microsphaera diffusa)	Georgia	Found during December in experimental plots in the greenhouse. (PDR 35: 221)
DELPHINIUM	Connecticut	No previous report of this disease in
Crown rot *	New York	the U. S. or abroad. New species
(Diplodina delphinii)	New Jersey	(Phytopath. 40: 615)
LUNARIA ANNUA		
Leaf spot ** (Alternaria brassicae)	California	Found in 1941 and reported in 1950 (PDR 34: 406)
RIBES AUREUM	New Mexico	Found in a small ornamental planting
Root rot **	Wyoming	near Mesilla Park. Additional un-
Fomes ribis	California	reported specimens found in Mycological Collections as follows: on R. aureum in Wyoming in 1917, on R. glutinosum, another ornamental species, in California in 1920. (PDR 34: 212)
TUBEROSE		
Botrytis spot and blight ** (Botrytis elliptica)	California	Found in commercial plantings grown for cut flowers and bulbs (PDR 35: 45)

Table 2. (Continued)

Host disease (Cause)	Where found	Remarks
PEANUT, COTTON, SOYBEAN		
Sting nematode ** (Belonolaimus gracilis)	Virginia	Survey indicated that sting nematode can cause serious damage to peanut crops. (Phytopath. 41: 29)
PEPPERMINT		
Root rot ** Typhula sp. (? T. itoana)	Oregon	Noted in Grand Island in early March. (PDR 34: 322)
CHINESE CHESTNUT		
Oak wilt ** Chalara quercina	Missouri	Isolated from five Chinese chestnut trees. (PDR 34: 291; 35: 28)
PLUM (Prunus angustifolia)		
Anthracnose ** (Glomerella cingulata)	Georgia	Reported in June 1950. (PDR 34: 352)

DISEASES OF CEREAL CROPS

Investigations carried out at the Wisconsin Agricultural Experiment Station in cooperation with the Division of Cereal Crops and Diseases on anthracnose (Colletotrichum graminicola) showed that the fungus is a high-temperature organism with optimal mycelial development at 28° C. Temperature had little effect upon the final severity of seedling disease of Sudan grass, but relatively low temperatures reduced seedling disease in rye. Host specificity was observed among different isolates. Isolates from the several hosts showed a wide range in pathogenicity under identical conditions. (Bruehl and Dickson. Tech. Bull. U. S. Dept. Agr. 1,005, 37 pp. 1950).

James A. Lyle reported incidence of diseases of small grains in Alabama in 1949-1950. Surveys of grain fields and of small grain variety tests were made at intervals during the fall, winter, and spring. He stated that in general oat diseases are much more prevalent in southern and central Alabama than in northern Alabama because of more favorable weather conditions for the growth of the pathogenic organisms during the growing season of the host. No variety of oats, at any of the different locations in the State, showed good resistance to any of the major diseases. Wheat as a winter grain crop is of minor importance in the State. Comparative susceptibility was shown of the different varieties to the three most serious diseases, i.e., powdery mildew (Erysiphe graminis tritici), leaf rust (Puccinia rubigo-vera tritici), and Septoria leaf blotch, at four different locations. (PDR 34: 318).

H. H. McKinney, from studies on pigment disorders in small grains, concluded that apart from the pigment disorders induced by parasites and possibly by viruses not yet discovered, cold injury to the leaves and nitrogen deficiency accounted for most of the trouble. Often low temperatures affect the plastids in certain varieties, and the leaf tissue gradually turns pale green and may die. Most of the anthocyanin type of pigmentation, especially common in certain oat varieties, appears to follow abnormally low temperatures, or excessive soil moisture conditions, or both. (PDR 34: 151).

R. W. Leukel summarized the results of seed treatment tests on oats and barley in 1950. Certain materials were tested with regard to their effect on germination and their effectiveness in controlling the loose and covered smuts of oats and the stripe-disease of barley. (PDR 34: 314).

J. E. Machacek summarized data on the effectiveness of different seed-treatment fungicides in the control of certain seed-borne diseases of wheat, oats, barley, and flax in cooperative experiments at 15 stations in Canada and 7 in the United States in 1950. (PDR 35: 146).

In Illinois, oat and wheat seed treatment experiments in 1950 were reported by Koehler and Bever. Special attention was given to dosage, storage before planting, and thoroughness of mixing. (PDR 34: 259).

AVENA SATIVA. OATS: In reporting on the occurrence and importance of oat diseases in Arkansas in the 1949-50 season, H. R. Rosen stated that Puccinia coronata, crown rust, was by far more common in 1950 than it had been during the past few years, causing considerable reduction in yield. Helminthosporium avenae, leaf spot, continued to be one of the most common of all oat diseases. (PDR 35: 149).

Erysiphe graminis, powdery mildew, has increased in North Carolina in the last few years. Helminthosporium avenae, leaf blotch, was the most serious disease on oats this past season in North Carolina. It was observed in fields throughout the State. A 10 percent reduction in grain yield was estimated. H. victoriae, Victoria blight, was first observed in North Carolina in the spring of 1949, in Hyde County, where it was present in only a few fields. The 1950 survey showed the disease not so severe as in 1949. Puccinia coronata, crown rust, was very prevalent in the fall in eastern North Carolina. Some fields were so badly damaged that the oats were plowed under. The 1950 spring crown rust infection was very light. (PDR 34: 392).

Puccinia graminis avenae race 7, oat stem rust, previously restricted principally to barberry areas, attacks Mindo, Bonda, Clinton, and other varieties of oats deriving their stem-rust resistance from White Tartar. It was found from Florida to Texas and northward to the Canadian boundary in 1950 and comprised almost one-half of the isolates in the United States. It is possible that this race can now maintain itself independently of barberries. (Stakman and Loegering. Phytopath. 41: 33).

HORDEUM VULGARE. BARLEY: Erysiphe graminis, powdery mildew, was very prevalent in the fall, but was not so severe in the spring in North Carolina. It was estimated that the average yield for the State was reduced about 5 percent. Puccinia hordei, leaf rust, which was present over the entire State, reduced the yield about 10 percent. Rhynchosporium secalis, scald, was less severe in 1950 than in 1949, but was found in every section of the State. The average loss was estimated at 2 percent. Ustilago nuda, loose smut, was present in most fields throughout the State, in trace amounts to 10 percent. (Moseman and Hebert. PDR 34: 392).

False-stripe (virus). H. H. McKinney reported that leaves of Chevron and Mars barley, showing false stripe in the nursery at St. Paul, Minnesota were sent to him in July and August 1950. Tests showed this virus to be seed-borne. Preliminary studies indicated that this virus probably is distinct from any previously described. The circumstantial evidence suggested rather strongly that the false-stripe and the chlorotic markings in the Chevalier barley plants collected by A. G. Johnson in 1913 were induced by this virus. (PDR 35: 48).

SORGHUM VULGARE. SORGHUM: Results of sorghum seed treatment tests at Beltsville, Maryland in 1950 were reported by R. W. Leukel (PDR 34: 342).

TRITICUM AESTIVUM. WHEAT: Ophiobolus graminis, take-all, caused slight to moderate damage in the southeastern quarter of Nebraska, according to J. E. Livingston. Even so, it was the most serious outbreak of take-all that he had seen in the State. A different type of root rot was found in many fields in the panhandle of western Nebraska resulting in a 10 to 20 percent reduction in stand. The cause of this root rot was not determined. Field observations over a period of years indicated that avoiding early planting and planting in a good firm seed bed are very desirable in avoiding this disease. (PDR 34: 232).

Puccinia spp., rust. According to Tervet and Cassell the development of small cyclone separators and their dual use both as collectors and disseminators of rust spores has facilitated the handling of uredospores and thus made the task of rust race identification easier. They described some methods that have proved successful in studies with cereal rusts. (Phytopath. 41: 286).

Puccinia graminis tritici race 15B, the most virulent race of stem rust ever found in North America and previously found almost exclusively near barberries, spread and multiplied spectacularly in 1950. It was found in 12 States from Texas to northernmost United States and comprised nearly one-fourth of all isolates. It attacked all hitherto stem-rust-resistant commercial varieties of durum and bread wheat and caused heavy damage to durums in northern Minnesota and North Dakota. It is possible that this dangerous race can now maintain itself independently of barberries. (Stakman and Loegering. Phytopath. 41: 33). In North Dakota the stem rust epidemic of 1950 was the most destructive since 1935 and losses were severe, according to W. E. Brentzel. Surveys showed that rust was widespread and in epidemic form in the eastern portion of the State. In the hard wheats the severity of infection ranged from a trace in the western part to as much as 80 to 90 percent in the eastern portion. The infection was particularly severe in the Red River Valley. Durum fields were damaged more severely

than the hard wheats, owing to the fact that they were somewhat later in maturing and are grown in the northeastern part of the State where the season is later. Red durum (D-5), notoriously known for its rust resistance and a variety which has resisted rust in all epidemics since 1914, rusted severely this year in North Dakota. The cause of the 1950 epidemic was two-fold. Seeding throughout the eastern part of the State was the latest on record, because of floods and a very late spring season. Harvesting was almost as late as seeding. The cool temperatures between these times were favorable for rust propagation. Perhaps the most important cause was the appearance of a race of rust uncommon or not heretofore known to occur in the Red River Valley. (PDR 34: 340)

Puccinia rubigo-vera tritici, leaf rust, was starting to build up on April 28, 1950, in Franklin County. According to R. S. Kirby this is one of the earliest dates in the spring that leaf rust has been observed in Pennsylvania. (PDR 34: 198). Moseman and Hebert reported that leaf rust caused an estimated loss of 10 percent in North Carolina. (PDR 34: 392)

Ustilago tritici, loose smut, was present in approximately 50 percent of the fields in Nebraska with as many as 5 percent of the heads affected in some fields. (J. E. Livingston, PDR 34: 232)

Mosaic (virus). J. R. Wallin reported on results of a survey for wheat mosaic in Kansas, Nebraska and Iowa. In Iowa a thirty-acre field showed 100 percent of the plants stunted, exhibiting mosaic symptoms. Some mosaic was found in Kansas and Nebraska. (PDR 34: 211)

ZEA MAYS. CORN: In Virginia, Curtis W. Roane reported that corn diseases were destructive in 1950 because of weather conditions favorable for their development and because of the general use of highly susceptible hybrids. Conditions were especially favorable for stalk rot development. It was estimated that stalk and ear rots would cost Virginia farmers 15 to 20 percent of their crop this season. (PDR 34: 394)

Bacterium stewartii, bacterial wilt or Stewart's disease, was reported on sweet corn in Connecticut, and in New York on Long Island and in the Hudson Valley as far north as Columbia County. Chupp stated that it had been shown many times that if the combined average temperatures of December, January, and February are less than 100° F., the disease will not be present, but if this sum is above 100°, and especially when it is above for two successive years, the disease may be important. (Letters to PDS Warning Service). In Illinois G. H. Boewe forecasted the prospects of bacterial wilt of corn for 1950. He stated that it would probably occur much farther north and be much more destructive in Illinois than it was in the summer of 1949. This forecast, the second to be made for Illinois, was based on the close relationship that appears to exist between the amount of disease which develops during the summer and the temperature of the preceding winter. In 1949, the development of both phases of the disease on sweet corn coincided very closely with the prediction made in the first forecast. Observations indicated that the leaf blight phase of the disease in field corn also can be predicted on the basis of winter temperatures. In many corn fields leaf blight was severe to very destructive in the south half of the State and light to moderately severe in north central Illinois in 1949. (PDR 34 (5): 155)

Helminthosporium turcicum, leaf blight, in 1950 occurred generally over Iowa, according to Vestal and Semeniuk. Leaf infection varied from a trace to 100 percent. Estimated yield reductions varied from a trace to 20 percent, the average being 1 percent. (PDR 35: 212) A preliminary report of results from some fungicide spray trials for control of Helminthosporium leaf blight of sweet corn was sent in by Warren N. Stoner. Some materials used in this experiment gave promise of effectiveness but further investigation seemed to be needed. (PDR 34: 312)

Puccinia sorghi, rust, according to J. R. Wallin, has been known for many years in this country, but without much damage in the corn belt. The disease became epiphytotic in 1950 in the North Central States. He quoted reports on the occurrence and severity of the rust in these States during 1950. In eight of the ten States reporting the 1950 weather conditions were mentioned as being unusually favorable to rust development. (PDR 35: 207)

DISEASES OF FORAGE AND COVER CROPS

CALAMAGROSTIS CANADENSIS. BLUEJOINT: S. Goto and J. W. Gibler described a nematode leaf gall found on bluejoint near St. Paul, Minnesota, in 1949. No spread from the original spot of infestation was observed in 1950. (PDR 35: 215)

CROTALARIA SPECTABILIS. SHOWY CROTALARIA: Charles M. Gates reported the presence of mosaic of C. spectabilis in southern Florida, as well as a modification of the mosaic disease designated as "little-leaf" disease (PDR 34: 203)

CYAMOPSIS TETRAGONOLOBA. GUAR: In Georgia during the summer of 1950, nursery plantings of guar were severely damaged by Alternaria leaf spot (Alternaria cucumerina) and white mold (Sclerotium rolfsii), according to E. S. Luttrell, who described the diseases. Observations indicated that both of these diseases may prove to be important factors in limiting the production of guar in Georgia as a green manure crop, or especially, as a seed crop. None of the 55 strains of guar, recent introductions from India, showed a satisfactory degree of resistance to either disease. (PDR 35: 166)

ERODIUM SPP. FILAREE: Red leaf (virus). Norman W. Frazier pointed out the symptoms of a virus disease of the yellows type that is of common occurrence on Erodium spp. in California. The virus was transmitted by three species of aphids. It was not transmitted by means of juice inoculation. (Phytopath. 41: 221)

FESTUCA SPP. FESCUE: Helminthosporium spp. K. W. Kreitlow and others reported the susceptibility of tall and meadow fescues to Helminthosporium infection. Data presented demonstrated that none of the strains of tall fescue tested was resistant to H. dictyoides. Differences in reaction to H. dictyoides among plants of meadow and tall fescues in both field and greenhouse suggested the possibility of isolating plants resistant to the disease. (PDR 34: 189)

H. dictyoides was found in a field of Kentucky 31 fescue in January near Baton Rouge, Louisiana. By mid-February following grazing the disease appeared to be less severe. Specimens received from other locations in Louisiana indicated that the disease is widespread on fescue. (J. G. Atkins. PDR 34: 157)

FESTUCA ELATIOR VAR. ARUNDINACEA. TALL FESCUE GRASS: In Georgia, diseases occurring on tall fescue grass were reported by E. S. Luttrell. Helminthosporium dictyoides, net blotch, occurred throughout the State and reached its peak in the late winter and early spring of 1949-50. Estimates of damage varied from a trace to 5 percent. Rhizoctonia leaf blotch (R. solani) was a serious disease on fescue throughout Georgia during the summer of 1949. During the drier summer of 1950 it was much less abundant. The highest estimate of damage was 5 percent. (PDR 35: 83)

LUPINUS ANGUSTIFOLIUS. BLUE LUPINE: Ascochyta gossypii, canker, was reported on blue lupine in Georgia by J. L. Weimer. This disease appeared to be limited largely to lupines in fields in which cotton had been growing. (PDR 35: 81)

Virus diseases. Weimer also reported that two virus diseases of blue lupine, seldom observed previously, were present in epiphytotic form in lupine fields at two locations in Georgia in the spring of 1949. In 1950 both diseases were present in numerous fields, but usually only an occasional plant was infected. A few observations and data were given. (PDR 34: 376)

MEDICAGO SATIVA. ALFALFA: In Virginia during the period April 11 to 13, alfalfa and clover fields were visited in six counties by Fenne and others. In most fields visited, injuries were noted on the roots of alfalfa plants, caused either by insects or Rhizoctonia spp. Considerable downy mildew (Peronospora trifoliorum) was observed. Blackstem (Ascochyta imperfecta) was abundant on the plants, causing both leaf spots and withering of the shoots, due to girdling. The stem nematode (Ditylenchus spp.) disease has continued to develop in Henrico County, where it was found in 1948 and 1949. Stemphylium botryosum, attacking both leaves and stems, is one of the big problems in growing alfalfa, according to Henderson and Smith. (PDR 34: 204)

Colletotrichum trifolii, anthracnose, in the wet summer of 1950 became widespread and locally destructive in the alfalfa nursery at Madison, Wisconsin according to F. R. Jones. In both the greenhouse and nursery certain selfed lines and crosses appeared to be far more susceptible than others. (PDR 34: 344)

Corynebacterium insidiosum, wilt. Valleau, in Kentucky, reported that specimens of alfalfa dying from wilt were received from Nicholas County. According to the report the majority of the plants were affected. This is the second report of alfalfa wilt in Kentucky in several years. (PDR 34: 214)

Diplodia gossypina is one of the causes of crown rot of alfalfa in Arizona, according to Bailey Sleeth. (PDR 35: 50)

Witches' broom (virus) disease is becoming an economic menace in the important Yuma Mesa alfalfa area of Arizona, according to Bailey Sleeth. In the Yuma Mesa 90 percent of the land is in alfalfa production. (PDR 35: 77)

Yellows. Increased incidence of alfalfa yellows in Virginia in 1950 was associated with unusual abundance of the three-cornered alfalfa hopper, according to S. B. Fenne and J. O. Rowell. The last outbreak in this State occurred in the summer of 1941. The insect is noticed especially following mild winters, such as were experienced for the past two years. (PDR 34: 344)

MELILOTUS SPP. SWEETCLOVER: Phytophthora cactorum, root rot, as reported by J. W. Gerdemann, is responsible for the loss of many sweetclover stands. In some sections of Illinois the growing of sweetclover has been discontinued because of this disease. Experiments in Illinois showed that occurrence of root rot is not affected by fertilizers added to the soil. It appears that the most promising control for this disease may be the development of resistant varieties. (PDR 34: 261)

SETARIA VIRIDIS. In tests made on common "weed grasses", it appeared probable that S. viridis in South Dakota provided a method of perpetuating wheat mosaic viruses between harvest and the emergence of volunteer and fall-sown wheat, by acting as a carry-over host. Winter wheat thus infected in the fall can harbor the viruses over winter. (John T. Slykhuis PDR 35: 221)

SOJA MAX. SOYBEAN: In Virginia, data presented by S. B. Fenne and W. C. White indicated that soybean seed treatment with Arasan increased germination. These results raised some questions for certification. Should such seed be eligible for certification, if percentage of germination meets certification requirements after treatment? (PDR 34: 206)
T. J. Nugent and others reported results of combined seed treatment and seed inoculation studies with soybeans in Virginia. The use of Arasan seed treatment significantly increased the stand counts of the soybeans in the test. Root nodulation was heavier on plants grown from seed which had been inoculated with Nitragin just prior to planting. (PDR 35: 82)
In North Carolina, during August 1950, leaf spot (Sclerotium rolfsii) was found on soybean leaves, and reported by Lehman and others. (PDR 35: 187)

TRIFOLIUM INCARNATUM. CRIMSON CLOVER: In Virginia, during the period of April 11 to 13, alfalfa and clover fields were visited in six counties by Fenne and others. Many fields of crimson clover were observed in the eastern part of the State, severely attacked by Sclerotinia trifoliorum. Three weeks of dry weather checked the development of stem rot and the severe losses expected did not occur generally. (PDR 34: 204)
Sclerotinia trifoliorum, crown rot, according to W. D. Valleau was severe on crimson clover over the State of Kentucky, probably because of a warm open winter with considerable rainfall. (PDR 34: 197)

TRIFOLIUM REPENS. LADINO CLOVER: Curvularia trifolii, leaf blight, was reported in North Carolina by S. G. Lehman. It developed rapidly during wet warm periods in fall, winter and spring. In areas along roadways or fences where the foliage is allowed to stand for long periods 20 to 25 percent of the leaves were diseased and a high proportion of them dead. (PDR 35: 79)

VICIA VILLOSA. HAIRY VETCH: Ascochyta sp., black stem, was present in all fields of hairy vetch observed in Kentucky. The vetch died out nearly completely in many fields. The winter was warm and wet, which undoubtedly contributed to the severity of the disease. (W. D. Valleau, PDR 34: 213)
Ascochyta pisi, blight, and Colletotrichum villosum, anthracnose, were reported by J. Lewis Allison and others as being destructive to the 1950 vetch seed crop in Tennessee. Rapid vegetative growth during the unusually warm winter months followed by a period of high rainfall during May and the first part of June contributed to the build up of the epiphytotics in 1950. (PDR 34: 321)

DISEASES OF FRUIT CROPS

The zinc deficiency situation in the western United States was reviewed by Wann and Thorne. The condition was present to a serious extent in all the major fruit- and nut-producing areas, and the cost of maintaining the crops in a healthy condition, usually by means of a dormant spray of zinc sulfate, was of economic significance, amounting in California to nearly $1,500,000 per annum. They pointed out that there is evidence of a gradual depletion of the available zinc in the

deeper soil strata under orchard trees, which is likely to lead to a considerable extension in the distribution of the deficiency in years to come. (Sci. Mon., N.Y. 70: 180-184)

CITRUS SPP. Cold spot -- a chlorosis of citrus leaves resembling virus ringspot -- has been described by L. Carl Knorr from Florida on orange leaves. That these chlorotic ringspots are caused by cold appears to be circumstantially true, but by what mechanism they arise still remains a question. (Citrus Magazine 12: 29-30, June 1950)

CITRUS AURANTIFOLIA. LIME: Results of a survey made to determine the amounts of lime bark disease and presumed virus disease in Dade County, Florida, and to attempt to correlate the presence of the virus disease to the severity of the Diplodia and Diaporthe bark rots, were reported by Gates and Soule. (PDR 34: 397)

CITRUS PARADISI X C. RETICULATA. TANGELO: J. F. L. Childs reported that a disease designated as "cachexia" disease, of unknown cause but resembling a virus disease, threatens the cultivation of the Orlando tangelo variety in Florida. The symptoms are described and the causal relationships discussed. (PDR 34: 295)

FRAGARIA SPP. STRAWBERRY: D. H. Scott and others reported tests indicating that it should not be difficult to originate strawberry varieties having resistance to both known strains of the red stele (Phytophthora fragariae) root disease fungus. (PDR 35: 134)

Virus diseases. Norman W. Frazier pointed out that strawberry species and varieties may differ markedly in their reaction to strawberry virus diseases. Investigations of strawberry viruses are greatly aided by the use of a test plant that may be readily infected with the different viruses and will clearly express symptoms. F. bracteata was found to be an excellent test plant, that showed marked sensitivity to infection, good differentiation of symptoms, and suitable adaptation to year-round greenhouse conditions. (PDR 35: 127)

P. W. Miller (p. 129) discussed the extent and distribution of virus in wild strawberries in Oregon in 1950. He stated that from the results of this study, it would appear that the presence of wild strawberries in the vicinity of cultivated fields will henceforth have to be taken into account in the general control program.

Miller and F. D. Aldrich (p. 131) reported that the Lindner staining procedure proved to be of limited value for the detection of strawberry viruses.

Yellows. E. P. Breakey and Leo Campbell described experiments, 1947-1950, demonstrating that strawberry plantings can be protected from spread of strawberry yellows by application of insecticides to control the strawberry aphid (Capitophorus fragaefolii). The yellows disease of the strawberry has become the limiting factor in the production of the Marshall variety in the Northwest. The expansion of the industry has been accompanied by a steady increase of the yellows disease. Roguing infected plants from new plantings as soon as the symptoms developed was found to be an essential part of the program. (PDR 35: 63)

MALUS SYLVESTRIS. APPLE. Botrytis sp., leaf spot. C. N. Clayton reported and described a leaf spot of apple, that was very common in many orchards in Wilkes and Alexander Counties, North Carolina in May 1950, due to a species of Botrytis. (PDR 35: 237)

Gymnosporangium juniperi-virginianae, cedar-apple rust, and black rot (Physalospora obtusa) in Connecticut, according to Rich and Stoddard were more prominent this year than in other seasons. (PDR 34: 240)

Stereum purpureum, silver streak, was found to be prevalent in apple orchards in the Spokane Valley and other parts of eastern Washington in 1923. The disease was observed again in the fall of 1950, the first time in a number of years. Symptoms were found in areas where trees had suffered considerable damage from severity of the 1948-1949 and 1949-1950 winters. (Sprague and Hord. PDR 34: 414)

Venturia inaequalis, scab. O. C. Boyd reported apple scab ascospores maturing abnormally early in Massachusetts. (PDR 34: 157). Heuberger, Poulos and Hood reported the earliest development of ascospores on record in Delaware. Ascospores were fully formed in some asci on overwintered leaves collected January 27 at Dover. On leaves collected at the same location on February 6, fully mature colored ascospores were found. January temperatures averaged 10.5° above normal and rainfall was abundant. (PDR 34: 71). Poulos and Heuberger reported a promising fungicide-plant food combination for control of apple scab. (PDR 34: 239)

In Illinois, H. W. Anderson reported that on February 2 he found a few perithecia showing several asci with mature ascospores. These were collected at Urbana, on Jonathan leaves. He stated that this early date consititutes a record for Illinois although in 1942 mature ascospores

were found on February 5. Weather conditions in 1942 were much like those in 1949-50 except that much higher maximums occurred in January 1950. (PDR 34: 71). Scab became serious on McIntosh apples in Connecticut in spite of a very early discharge of ascospores this spring. This condition was caused by a period of warm, wet weather during the last half of May. (Rich and Stoddard. PDR 34: 240). M. C. Richards reported New Hampshire experience with Sulfur-NuGreen combinations for the control of scab. (PDR 34: 299)

PRUNUS. SPP. CHERRY: Ringspot (virus). The stunting effect of latent ringspot virus infection in cherry trees was evident from Oregon experiments reported by J. A. Milbrath. (PDR 34: 374)

PRUNUS ANGUSTIFOLIA. WILD PLUM: Phony peach disease (virus). H. L. Bruer and others reported results of a survey of incidence of phony disease in wild Prunus. In February 1950 a survey was begun of wild Prunus in the southeastern States using the technique of chemically testing stems. P. angustifolia was the predominant species in the area surveyed, therefore the greater number of twigs tested were of that species. Tests were made and material secured from 31 counties in Alabama, Georgia, Mississippi, and South Carolina. Positive reactions were secured from at least one location in 23 of these counties. (PDR 35: 186)

PRUNUS AVIUM. SWEET CHERRY: Buckskin (virus). Nichols and Nyland reported buckskin fruit symptoms on sweet cherry trees on Mahaleb stock. (PDR 35: 32)

Dixie rusty mottle (virus) according to B. L. Richards and others is the most serious of three rusty mottle virus diseases found in Utah. The disease is known to be present only in Washington County, where it is also destructive to peach. Because of the destructive nature of the disease, also because of the ease with which the causal virus can be transmitted, Dixie rusty mottle must be considered a definite threat to the entire cherry industry of Utah. In the cherry the Dixie rusty mottle virus is a killer. (Farm and Home Science 11 (2): 36-37, June 1950)

Winter injury. Conditions accompanying the severe winter injury suffered by sweet cherries in Montana during 1948-49 and 1949-50 were discussed by H. E. Morris and M. M. Afanasiev. The injury manifested itself mainly in sun scald or in flower bud injuries. (PDR 35: 192)

Virus diseases. M. M. Afanasiev and H. E. Morris reported on occurrence of virus diseases in Montana in 1950. Surveys showed that the following virus diseases were found: mottle leaf, ring-spot complex, rasp and twisted leaf. These viroses were present only in traces, with the exception of a rather high percentage of ring-spot complex on May Duke and Stark Golden. Little cherry virosis of sweet cherry has not been reported from Montana. In the spring of 1950 a large number of trees in one orchard showed symptoms similar to a virosis called Lambert mottle. Blotchy mottling was found in practically all varieties of cherries. Traces of crinkle and deep suture were found on several varieties of cherries. (PDR 35: 191)

PRUNUS CERASUS. SOUR CHERRY: Western X-disease (virus). Zeller and Milbrath reported the recovery of western X-disease of peach from Montmorency cherry. Experiments indicated that western X-disease of peach, red-leafed chokecherry, and buckskin of sweet and sour cherries as they occur in Wasco County, Oregon, are caused by the same virus. (Phytopath. 40: 707)

PRUNUS PERSICA: PEACH: Diaporthe eres, constriction disease, is of major importance on the Eastern Shore of Maryland in certain seasons favorable for the disease, according to L. O. Weaver. The disease has not been controlled either by spraying or by pruning. The susceptible Golden Jubilee variety was removed from the orchards by the growers during 1950. (PDR 35: 144)

Meloidogyne incognita and M. javanica, root knot nematodes. According to Leon Havis and others the problem or root knot nematode damage to peach tree roots is one of the most serious faced by growers and nurserymen in the more southern States. They give a brief report of a detailed study of the relative susceptibliity of some peach rootstocks to the two principal species of peach nematodes. The great variability in resistance to the two nematode species shown by the hybrid seedling stock was encouraging. It is thought that through modern breeding methods nematode resistant stocks giving good orchard trees can be developed. (PDR 34: 74)

Monilinia fructicola, brown rot. In Illinois experiments in 1949 and 1950 reported by Dwight Powell showed that sprays containing Phygon XL were superior to other treatments in reducing blossom blight infection on peaches. By increasing control of such infection during the

blossom period it was possible to retard the rate of development of the preharvest fruit infection. (PDR 35: 76)

Phony peach (virus) control has been complicated by the spread of the disease on wild Prunus. According to Bruer and others the disease is apparently more prevalent in wild Prunus in those areas where records of orchard inspections demonstrate that there is higher incidence in peach. Apparently the factors that favor the rapid spread and development of the disease in one host are also favorable for its development in the alternate host. (PDR 35: 186)

Ring pox (virus). Experimental transmission of the apricot ring pox virus to peach and plum was reported by Austin O. Simonds. (PDR 35: 189)

Yellow leaf roll (? virus). In Yuba and Sutter Counties, California, Nyland and Schlocker reported a disease they are calling yellow leaf roll. There seems little doubt that the causal agent is a virus. The disease has spread rapidly during the last three years. In one block of about 900 trees, 8 diseased trees were found in 1948, 25 additional trees in 1949, and 80 additional trees in 1950. (PDR 35: 33).

PYRUS COMMUNIS. PEAR: Isopropanol, a constituent of a glyoxalidine fungicide, hastened the ripening of Bartlett pears after harvest according to evidence reported by E. E. Wilson. (PDR 35: 38)

RUBUS SPP. CANE FRUITS: Edward K. Vaughan and others reported the results of a survey of cane fruit diseases which was conducted in the Pacific Northwest during June 1950. The purpose of the survey was to determine what virus diseases were present in the area, where and on what varieties they were the most prevalent, and the relative severity of each. Observations were also made for diseases of pathogenic and physiologic origin. (PDR 35: 34)

VACCINIUM SPP. BLUEBERRY: Physalospora corticis, stem canker. Demaree and Morrow reported that stem canker in blueberries is southern in its adaptation and distribution. They discussed relative resistance of some blueberry varieties and selections to stem canker in North Carolina. There was no immediate problem concerning stem canker resistance in the rabbiteye blueberry as all standard varieties were either immune or highly resistant. (PDR 35: 136)

VITIS SPP. GRAPE: Cryptosporella viticola, dead arm. Wm. B. Hewitt reported results of spray experiments for control of this disease in California. The severity of the disease was only moderate in 1950. DN-289 appeared to have considerable promise as a spray control for the spring infections of dead-arm and was also less hazardous than sodium arsenite. (PDR 35: 142)

Mosaic (virus). Hewitt stated that vine mosaic (virus) was becoming increasingly prevalent in young vineyards in California. Spread appeared to occur through buds or scion wood from affected vines. The best method of preventing spread consisted in selecting, propagating, and planting only clean, healthy stock. (Bull. Dept. Agr. Calif. 39, 2, p. 61, 1950)

DISEASES OF NUT CROPS

CARYA ILLINOENSIS. PECAN: Articularia quercina var. minor was collected on pecan at the Brownwood Station, Texas, in 1950. The first collection in Texas was made in 1933. The fungus was collected in Mississippi in 1920, on Quercus sp. (John R. Cole. PDR 34: 414)

P. W. Miller reported on nut diseases in Oregon in 1950:

CORYLUS SP. FILBERT: Phyllactinia corylea, mildew, was of no economic importance, since it did not make its appearance until late in the season.

Xanthomonas corylina, bacterial blight, was widely distributed in 1950. It was most prevalent in young orchards, where it caused the death of a number of young trees.

Brown-stain (non-parasitic) was present in limited amounts in some filbert orchards in western Oregon in 1950. The estimated average crop loss was 2 percent.

JUGLANS REGIA. PERSIAN WALNUT: Armillaria mellea, mushroom root rot, caused the death of some Persian walnuts and of a limited number of Franquette walnuts grafted on Hind's black walnut rootstock (Juglans hindsii). This finding is noteworthy as this rootstock is considered to be highly resistant to this disease.

Blackline (girdle) of grafted walnuts (non-parasitic) was responsible for the death of more grafted Franquette walnut trees in 1950 in Oregon than any other disease.

Leaf scorch (non-parasitic) was widely distributed in the Pacific Northwest in 1950, occurring in many Persian walnut orchards in Oregon. (PDR 35: 145)

DISEASES OF ORNAMENTALS

R. A. Jehle and others listed spot anthracnoses known to occur in Maryland and their distribution by counties. (PDR 35: 194)

A. C. Tarjan presented observations on nematodes associated with decline or ornamental plantings, and concluded that the staggering weight of evidence must eventually force realization of the seriousness of the problem and stimulate much needed research. (PDR 35: 217)

Studies on vectors, hosts and properties of dahlia-mosaic virus were reported by Philip Brierley and Floyd F. Smith. Sanvitalia procumbens, Verbesina encelioides, and Zinnia elegans can be infected with dahlia-mosaic virus, developing symptoms similar to those expressed by dahlia. Coreopsis douglasii developed no symptoms when inoculated, but dahlia-mosaic virus was recovered. (PDR 34: 369)

ACHILLEA SPP. YARROW: Agrobacterium tumefaciens, crown gall, was observed on ornamental types of yarrow imported from out of the State of California. (PDR 35: 42)

ANTIRRHINUM MAJUS. SNAPDRAGON: Oidium sp. of the Erysiphe cichoracearum type, powdery mildew. MacLean and Baker reported occurrence of powdery mildew on outdoor snapdragons in home gardens in the Los Angeles and Berkeley areas under humid and shaded conditions. Probably the fungus has recently been introduced on greenhouse planting stock into these two areas of California and its spread has so far been limited to these areas. (PDR 34: 346)

CAMELLIA SPP. CAMELLIA: The Bureau of Entomology and Plant Quarantine reported the results of the cooperative Federal-State camellia flower blight survey from December 1949 to March 1950. The States included Alabama, Florida, Georgia, Louisiana, Mississippi, New York, North Carolina, South Carolina, Tennessee and Texas. The survey disclosed that while most of the trade in camellia plants in the Southern States and Texas is local within that group of States, a considerable number of plants, shipped with soil, have been obtained by growers in these States from California for a number of years. Positive determinations were made from specimens collected in North Carolina and Georgia. (PDR 34: 264)

CHRYSANTHEMUM MAXIMUM. ESTHER READ DAISY: K. F. Baker reported that fasciation disease (Corynebacterium fascians) occurs on many ornamentals in California, but causes losses of economic importance only to Esther Read daisy. It was first observed on this host in various localities in southern California in 1945 on plants growing in irrigation ditches, and it reduced greatly the cut-flower production. The disease is now generally established in the southern part of the State. In March 1950, it was reported to be destructive in a commercial planting in San Mateo County. A list (compiled from the literature) is presented of the host range of the pathogen, consisting of 34 genera in 21 families. (PDR 34: 121)

Septoria leucanthemi, Septoria leaf spot, on Esther Read daisies has been a limiting factor in the production of this crop in the cool, foggy, coastal areas of San Mateo County, in California. Growers estimated that losses from this disease range from 15 to 50 percent of the crop, depending upon the weather during the growing season. Symptoms and results of control tests were discussed. (N. A. MacLean, Florists' Review 107: 35, Nov. 23, 1950)

CHRYSANTHEMUM MORIFOLIUM. CHRYSANTHEMUM: Septoria chrysanthemi, Septoria leaf spot, has been reported as serious in the eastern United States, but is usually of minor importance on commercial outdoor chrysanthemums in California. The only recorded severe outbreak was in February 1950, when the disease was found to be serious on chrysanthemum cuttings under cloth in the Redwood City area of San Mateo County.

DIANTHUS CARYOPHYLLUS. CARNATION: Peronospora dianthicola was reported by Gardner and Yarwood in a nursery at Colma, California. This disease has occurred every year since 1946. (PDR 34: 270)

Fusarium wilt. Stoddard and Dimond stated that the chemotherapeutic approach to the control of plant diseases is a rapidly advancing realm of plant pathology. They reported on the chemotherapeutic control of Fusarium wilt of carnations. (Phytopath. 41: 337)

ERICA SPP. HEATHER: Chlorosis. Spraying with ferrous sulfate controlled chlorosis in commercial plantings of heather in California. (Sciaroni and Zentmyer. PDR 34: 402)

GLADIOLUS SPP. GLADIOLUS: Fusarium orthoceras var. gladoli, corm rot and yellows is the most destructive disease of gladioli, according to Robert O. Magie, who stated that this disease destroyed approximately two million dollars' worth of gladiolus flower spikes and corms during the 1949 to 1950 season in Florida. This fungus exists in many strains. Florida probably harbors the most virulent strains, because corms are shipped there by the million from many States and foreign countries. Research has shown that the Fusarium disease can be controlled economically. Magie pointed out the most successful steps in a successful control program. (The Florists' Rev. 106, no. 2738; 28-30. May 1950). Results of tests for gladiolus dry rot, (Stromatinia gladioli), control in western Washington in 1950 were reported by Charles J. Gould. Mersolite definitely appeared to be promising. (PDR 35: 109)

LILIUM LONGIFLORUM: Frank P. McWhorter and C. J. Anderson reported diseases found in Croft lily plantings in Oregon in 1949-50. The principal diseases of L. longiflorum continued to be scale tip rot (Cylindrocarpon radicicola), Botrytis blight (Botrytis cinerea), scorch (physiological), dieback and bunchy top (Aphelenchoides olesistus), streak-fleck complex (virus), and Colletotrichum scale rot. Meadow nematodes (Pratylenchus spp.) were present in one California and four Oregon locations, and appeared to be the most destructive pest that has occurred in these lilies. (PDR 35: 106)

ORCHIDACEAE. ORCHIDS: Ark and Starr described three bacterial diseases, Phytomonas cattleyae, Erwinia cypripedii and an unidentified species of bacterium of orchids, and their control in central California. They sometimes cause considerable losses. (PDR 35:42)

PEPEROMIA SPP. PEPEROMIA: Phytophthora palmivora, rot, was found in 1950 on Peperomia plants in California. In one greenhouse 50 percent of the plants were affected by the disease. Peperomia plants are extensively propagated by nurserymen in the San Francisco Bay area. The disease was controlled by using cuttings from healthy plants and planting them in pasteurized soil mixture. (Ark and DeWolfe. PDR 35: 46)

DISEASES OF SPECIAL CROPS

ARACHIS HYPOGAEA. PEANUT: Sclerotium rolfsii, stem and pod rot, in New Mexico according to P. J. Leyendecker, Jr. accounted for a 20 to 30 percent reduction in yield in the Portales area. Approximately 7,000 acres were involved, with a cash loss to the growers of about $200,000. The disease was aggravated by high soil moisture and cool soil temperatures during July. (PDR 35: 168)

CARTHAMUS TINCTORIUS. SAFFLOWER: Alternaria carthami, leaf spot, in 1950, was of minor importance in Colorado and western Nebraska, but was prevalent in experimental plantings in central and eastern Nebraska, Kansas and Oklahoma, and Texas under more humid conditions. (C. A. Thomas. PDR 34: 391)

Donald C. Erwin reported that the cause of a root rot of safflower occurring in Nebraska, had been identified as Phytophthora drechsleri. A high percentage of infection was readily obtained in greenhouse pathogenicity tests. Some varieties have been reported to be resistant to root rot. No other control is known. Since the disease occurs at high temperatures, early planting and the limitation of irrigation to periods when the soil temperature is at a minimum, preferably 15° to 17° C., or lower, might be effective (PDR 34: 306). Thomas reported this root rot was very destructive in southern California, and that it was observed in Arizona, Oklahoma, Texas, Kansas, Colorado and Nebraska.

Puccinia carthami, rust, was much more prevalent this year in Colorado and Nebraska tha in 1949. A few plantings affected with rust were observed in Kansas and California. (C. A. Thomas. PDR 34: 391)

GOSSYPIUM SPP. COTTON: Ascochyta gossypii, Ascochyta seedling blight, became general in the northern third of Alabama in 1950, according to A. L. Smith. In DeKalb County many stands were reduced to between 50 and 70 percent. Frequent local showers and prolonged cool weather in this area were responsible for the epidemic. Evidence obtained indicated that the disease is seed-borne. All 25 varieties of cotton tested proved susceptible in the seedling

stage. The author suggested "wet weather blight" as a common name for the disease. (PDR 34: 233). According to Aycock and Arndt infection of young cotton plants by A. gossypii resulted in extensive losses for the fourth consecutive year in localized areas of South Carolina. The losses from boll rots in South Carolina in 1950 that were not associated with insects were relatively small as compared with those for 1949, when losses from the State as a whole averaged above 30 percent. The comparative weather records for 1949 and 1950 would seem to indicate that the amount of boll rot in these two years was related to the amount of rainfall in August rather than to that in July. About 80 percent of the bolls injured by insects and fungi were completely decayed. Most of the bolls were covered with the mycelia of Diplodia gossypina. (PDR 35: 204)

Verticillium albo-atrum, wilt, was found in several fields in Caddo, Bossier, and East Carroll Parishes, Louisiana, during July and August, infection ranging from 5 to 30 percent. This disease is now known to occur in three of the important cotton producing parishes of the State. (PDR 34: 353) This wilt was unusually severe during the 1950 season in the Mesilla Valley of New Mexico and El Paso Valley of Texas. Losses vary from year to year depending upon the time of the season when soil temperatures become favorable for disease expression. In the 1950 season the disease appeared early, in mid-July. (P. J. Leyendecker, Jr. PDR 35: 169) According to work at the Arizona Agricultural Experiment Station and reported by Ross M. Allen cotton seeds inoculated with the Verticillium become infected and carry the fungus over extended periods of time. (PDR 35: 11)

The factors contributing to Verticillium wilt of cotton in the Southwest have been in question for a number of years, according to Blank and Leyendecker. They showed data which demonstrated the potentialities of wilt-infected stalks as a means of spreading wilt to areas previously free from the disease. (PDR 35: 10)

MENTHA SPP. MINT: Puccinia menthae, rust, caused considerable damage to spearmint in the Midwest and many growers cut the herb early to avoid losses from leaf fall. Reports from Oregon indicated that peppermint was severely damaged by rust in that State (C. A. Thomas. PDR 34: 391). Row plantings of Scotch spearmint were dusted in 1948, 1949 and 1950 for the control of rust. In Michigan, plantings of peppermint adjacent to rusted spearmints remained rust-free. Control plants were moderately rusted in 1948, rust-free in 1949, and killed by the disease in 1950. Under severe conditions in 1950 Fermate gave protection much superior to that of other materials. Actidione and tribasic copper sulfate-zinc dusts are most likely to be useful in mint rust control. (Ray Nelson. Phytopath. 41: 27).

Verticillium albo-atrum var. menthae, wilt, was unusually severe on peppermint in Indiana and Michigan. Spearmint was also affected. Weather conditions were particularly favorable for heavy infection and development of the disease. (C. A. Thomas. PDR 34: 392)

NICOTIANA TABACUM. TOBACCO: Wilbert A. Jenkins described the symptoms of a seedling root rot-complex, associated with nematodes, causing tobacco plant bed failures in Pittsylvania County, Virginia. He doubts that the disease is a new one. (PDR 35: 177)

Peronospora tabacina, blue mold. Because of the mild, late outbreak of blue mold in 1949, and therefore limited carryover, Valleau and others in Kentucky predicted to farmers and manufacturers of ferbam that there would be an extremely mild outbreak in 1950, unless there was a blow-in of spores from the Southeast. There was some infection by June 9, but no injury was caused. Valleau stated that there should be no blue mold in Kentucky in 1951 unless there is a blow-in from the Southeast, causing early infection over Tennessee and Kentucky. Spores have blown in from the Southeast only twice, in 1937 and 1945, in the 20 years the disease has been established in the East (PDR 34: 210). In the final summary of the plant disease warning service (PDR Suppl. 197: 559), Miller and O'Brien stated that severe tobacco blue mold was reported in certain localities, but that the disease on the whole was relatively mild, despite the general distribution throughout the tobacco-growing areas. Fungicides used indicated the preference for Fermate.

Pseudomonas tabaci, wildfire. W. D. Valleau, in Kentucky, stated that probably the worst outbreak of wildfire experienced in the State in over 30 years was associated with an unusually wet May. Since Bordeaux did not give good control, it was concluded that the time of plowing the beds was the explanation. The wildfire bacteria live normally on the roots of many pasture plants and weeds. When the beds are plowed in the fall the roots die and the bacteria finally disappear from the soil, in the absence of live roots. When the sod is turned up in late winter and the bed is moderately burned the bacteria are probably abundant a couple of inches down. While wildfire was common, angular leaf spot (P. angulata) was rare. (PDR 34: 210)

Tobacco-mosaic virus. Francis O. Holmes furnished evidence to support the hypothesis that

tobacco-mosaic virus was of New World origin. (Phytopath. 41: 341)

RICINUS COMMUNIS. CASTOR BEAN: Alternaria ricini, capsule mold. Unusually wet summer weather in Oklahoma and northern Texas resulted in considerable capsule mold.

Plants in an experimental planting at Shafter, California were affected with what was tentatively identified as Verticillium wilt. The soil was known to be heavily infested with Verticillium albo-atrum which causes cotton wilt. (C. A. Thomas. PDR 34: 391)

RUMEX HYMENOSEPALUS. CANAIGRE: Meloidogyne incognita, root-knot nematode, infection is described on canaigre roots, as observed in Arizona, by Reynolds and Sleeth. They state that it is not known to what extent root-knot nematodes will reduce yields or impair the roots for processing. (PDR 35: 9)

Ovularia canaegricola, leaf spot, caused serious damage to experimental plantings of canaigre at Chandler Heights, Arizona, according to L. M. Pultz. The spread of the disease is being watched because of the possible effect of the leaf spot on root production in the plants. (PDR 34: 157). This disease is described and illustrated by J. G. Brown and Alice M. Boyle. Since it is spread by aerial conidiospores, they suggest that spraying the plant with Bordeaux mixture or other good fungicide should protect against Ovularia leaf spot. (PDR 34: 178)

SACCHARUM OFFICINARUM. SUGARCANE: Cercospora longipes, brown spot, a leaf disease, according to E. V. Abbott has been present in Louisiana for many years, but until the release and widespread growing of the susceptible variety, C.P. 36/105, it had not been of commercial importance owing to the resistance of the varieties previously grown. During the crop season of 1950 a combination of conditions, such as temperature and field practices, favored the disease so that it developed to rather severe proportions on C. P. 36/105 in several parts of the sugarcane district of the State. Surveys showed brown spot to be commonly present on this variety in the southeastern parishes. It was found to be prevalent in St. Mary, Iberia and St. Martin Parishes. It was observed on one farm in Lafayette Parish and was not found in Vermillion Parish. Abbot discussed the importance and losses caused by the disease and its control. (Sugar Bull. 29: 134, Feb. 1, 1951)

SESAMUM ORIENTALE. SESAME: Cercospora sesami, has been observed in Maryland, South Carolina and Texas. The organism is known to be seed-borne.

Fusarium wilt of sesame was found at Charlotte Court House, Virginia in 1950, according to Sears and Wingard. (PDR 35: 173)

Pseudomonas sesami, bacterial leaf spot, appeared to cause one of the most serious diseases of sesame. The disease has been observed from Maryland to Texas. Late plantings at Chillicothe, Texas were a complete loss from this disease. (C. A. Thomas. PDR 34: 392)

ZOSTERA MARINA. EELGRASS: A history of the wasting disease of eelgrass on the Atlantic Coast, with known facts about the epidemic, which if measured by its effect on the host plant is the most devastating in the history of plant pathology, and speculation as to the factors involved was reported by Neil E. Stevens, Hazel R. Ellis and Russell B. Stevens. (PDR 34: 357)

DISEASES OF TREES

David M. Marsden analyzed Massachusetts precipitation data since 1928, with reference to tree injury. (PDR 34: 400)

CASTANEA SPP. CHESTNUT: Bowen S. Crandall, in reviewing the distribution and importance of the chestnut root rot Phytophthoras (P. cinnamomi and P. cambivora), stated that it would appear that P. cinnamomi is one of the world's major plant killers. A monographic study of this species is certainly indicated since its wide distribution makes it probable that a historical study of its movements and distribution on plant groups would aid considerably in the understanding of the modes of distribution of plant pathogens which, unlike many, do not possess spore forms easily carried by various means. Many inferences could probably be drawn by a study of its known hosts and their resistant or immune relatives. (PDR 34: 195)

CASTANEA MOLLISSIMA. CHINESE CHESTNUT: Chalara quercina, oak wilt. T. W. Bretz reported that the pathogenicity of the oak wilt fungus to Chinese chestnut was demonstrated by experiments. Although the number of trees involved in this preliminary work was small the

results indicated that the oak wilt fungus was a virulent pathogen in the C. mollissima accessions tested and that interspecies transfer of the fungus from oak to chestnut may occur. (PDR 35: 28) The fungus was first isolated from 5 C. mollissima trees growing in a plantation in Missouri. (Bretz and Long. PDR 34: 291)

CORNUS FLORIDA. FLOWERING DOGWOOD: Ascochyta cornicola, leaf disease, that causes spots and shriveling of entire leaves of dogwood, has been epidemic through the mountain areas of Virginia and North Carolina in 1950. This same disease was epidemic in 1942 and had not been conspicuous enough to be noticed between those years. (George P. Hepting. PDR 34: 227)

Elsinoë corni, spot anthracnose. Jehle and Jenkins reported further observations on spot anthracnose in Worcester County, Maryland and Lynchburg, Panorama, and Madison, Virginia in 1950. (PDR 34: 225)

On July 6, 1950, leaves of dogwood, heavily infected with spot anthracnose, were collected at Trail's Cabin, Blue Ridge Parkway, near Floyd, Virginia, according to George P. Hepting. The Superintendent of the Parkway wrote that approximately 50 percent of the trees in the Parkway south of Roanoke were infected and on Section 1-U it was epidemic (PDR 34: 227)

PINUS PONDEROSA SCOPULORUM. PONDEROSA PINE: Cronartium comandrae, rust. According to Willis W. Wagener a severe cold spell practically eliminated comandra rust in experimental ponderosa pine plantations in Idaho. (PDR 34: 193)

PLATANUS OCCIDENTALIS. SYCAMORE: In Georgia, G. E. Thompson described a dieback of sycamore, which has been observed during the past two summers. Diplodia natalensis was isolated from the diseased wood. Inoculations were made on small trees, and within a few weeks all inoculations showed infection. (PDR 35: 30)

QUERCUS SPP. OAK: Marvin E. Fowler reported methods and results of the oak wilt (Chalara quercina) surveys in 1950, together with the known distribution of the disease in the United States. In 1950 willow oak (Quercus phellos) and southern red oak (Q. rubra) were found for the first time to be naturally infected. No native oak species is known that is immune to this disease. (PDR 35: 112)

With the discovery of extensive natural root grafting and of passage by the fungus from tree to tree through such grafts, local control of oak wilt in certain Wisconsin areas was accomplished Immediate removal of each wilting tree retarded the disease in 11 out of 18 eradication plots. There has been developed a tractor drawn knife that moves 100 feet per minute, and cuts roots to a depth of 36 inches. This cutter was used in 12 plots in 1950. (Kuntz and Riker. Phytopath. 41: 23)

Irpex mollis, canker. Elmer R. Roth described cankers and decay of oak trees associated with this fungus. Loss from I. mollis was believed to be of minor economic importance. Apparently there is no relation between tree vigor and the occurrence of I. mollis cankers, since they were found on both slow growing and fast growing oaks on both good and poor sites. Cankers are fairly common in the vicinity of Walhalla, South Carolina and in adjacent areas in Georgia. (PDR 34: 347) For oak wilt in Pennsylvania and Illinois see Table 1.

QUERCUS ALBA: WHITE OAK: Gnomonia veneta, anthracnose. Charles L. Fergus reported an epiphytotic of white oak anthracnose in Pennsylvania in 1950. No specific data are available for the severity of anthracnose on white oaks under forest conditions, but causal observations showed a considerable amount. Meteorological data for State College were examined in an attempt to correlate weather conditions and the recorded epiphytotics of 1944 and 1950. The weather conditions which prevailed in both 1944 and 1950 and which appear to have determined the occurrence of severe anthracnose were: (1) an open winter with temperatures of the winter months higher than normal, (2) a delayed spring with deep snow cover in March and temperatures of March and April lower than normal, (3) moderate precipitation recorded for a large number of days, and (4) low percentage of effective sunshine hours possible. (PDR 34: 292)

ULMUS AMERICANA. AMERICAN ELM: Zonate canker (virus) according to Swingle and Bretz, is a previously undescribed disease of Ulmus americana that is transmissible by grafting and apparently of virus nature. Attempted transmission of the zonate canker virus by leaf inoculations failed, but some transmission attempts other than by common grafting techniques were successful. The zonate canker virus has been found to occur in trees in New Jersey, Ohio

and Missouri. It has been found alone and in combination with the elm mosaic virus. (Phytopath. 40: 1018)

DISEASES OF VEGETABLE CROPS

Warren N. Stoner listed some diseases which caused major economic losses in vegetables in the Everglades region of Florida during the 1949-1950 season. (PDR 35: 170)

ALLIUM ASCALONICUM. SHALLOT: Peronospora destructor, downy mildew. In Louisiana, E. C. Tims reported that in the last four years, 1947-1950 inclusive, there have been three very destructive epidemics. The disease had caused such serious losses that some farmers who had grown onions for many years had given up the crop entirely. For many years mildew was observed occasionally on shallot, but never developed in epidemic proportions. During the past five years the disease has become a major problem for shallot growers especially along the Mississippi River and Bayou Lafourche sections. In 1950, mildew occurred in January on both shallots and onions. The losses from shallot in 1950 were probably the heaviest on record. Many fields were a total loss. Mildew appeared in the Angola shallot producing area for the first time in 1950. It was also very destructive on onions in 1950. The onion seed crop was almost a total failure. The final solution to this problem in Louisiana seems to be the development of resistant varieties. (PDR 34: 380)

ALLIUM CEPA. ONION: Peronospora destructor, downy mildew. In Connecticut, Rich and Stoddard reported that on June 21 a half-acre of onions being grown for sets was found to be 100 percent infected with downy mildew. The field had been used repeatedly for growing onions. The leaves were completely gone, the stalks were severely infected, and there was abundant conidial production. This was the first Connecticut report of such a heavy infestation of this disease in the past eleven years. See also under A. ascalonicum. (PDR 34: 240)

Urocystis cepulae, smut. In Cook County, Illinois, the pelleting of bulb-onion seed with Arasan (50% thiram, nonwettable in water) has replaced almost entirely the formaldehyde-drip treatment as a control for smut (Urocystis cepulae) according to M. B. Linn. From 95 to 100 percent of the plants from untreated seed were killed in many Cook County fields. (PDR 35: 94)

A. G. Newhall and W. W. Gunkel described a duster used in 1950 for row application of fungicides and insecticides at the time of sowing seeds. The method and equipment was used in New York in the control of onion smut and maggot. Much interest has been aroused since this method may lend itself to the control of other seedling diseases and insect pests of a wide variety of crops. (PDR 35: 219)

APIUM GRAVEOLENS. CELERY: Cephalosporium sp., brown spot. Occurrence of this brown spot disease on stalks of celery in New York apparently is correlated with high seasonal temperatures, according to observations reported by Ralph Segall. The temperature during the 1950 season was slightly below normal, and the disease was not found. (PDR 35:.164)

CUCURBITS. CUCUMBER, MELON, SQUASH: Pseudomonas lachrymans, angular leaf spot. F. S. Beecher and S. P. Doolittle gave evidence indicative of insect transmission of angular leaf spot. (PDR 34: 382). In another article they reported control of angular leaf spot with a copper fungicide. (PDR 34: 383)

D. E. Ellis, in reporting occurrence and control of some noteworthy diseases in North Carolina, stated that anthracnose (Colletotrichum lagenarium) caused as much or more damage to cucumber in 1950 as did downy mildew.

Pseudoperonospora cubensis, downy mildew, developed more slowly on cucumber in North Carolina than it did on muskmelon. It was thought that a new physiological race of the fungus might be present in 1950. (PDR 35: 91). G. K. Parris reported that it has been found possible to preserve the viability and infective power of conidia of P. cubensis, on detached squash leaves, for six months. (PDR 35: 52). In the summary of the plant disease warning service for 1950, Miller and O'Brien stated that cucurbit downy mildew occurred along the Atlantic Coast seaboard and in the southern part of Texas. Losses were reduced, since the disease appeared later than normal when the crop was mature or nearing maturity.

Mosaic (virus). In California the response of the mosaic-infected cantaloupes to nitrogen fertilizer appeared to be an increase in size of fruit, and reduction in number of slick fruits. No increase in the number of fruits set, total soluble solids of the fruits, nor retardation in the rate of maturity was apparent with the use of more than 60 pounds of nitrogen per acre. (F. W. Zink

and G. N. Davis, PDR 34: 371). Mosaic was unusually severe in experimental plantings of cucumber and summer squash in Duplin County, North Carolina in the fall of 1950. (PDR 35: 91)

Incidence of some vegetable crop diseases in Massachusetts was reported by O. C. Boyd. He stated that in no previous season have the cucurbit viruses been so damaging. It was doubtful if a single planting of cucumbers escaped mosaic, and some fields, the earlier plantings in particular, were almost totally destroyed. Even the fields and gardens that were thoroughly dusted or sprayed suffered moderately to severely. Thrips were abnormally numerous this year. (PDR 34: 339)

Preliminary data toward identification of three viruses or virus strains infectious to cucurbits, collected in central Florida in 1950 were reported by C. W. Anderson. (PDR 35: 233)

IPOMOEA BATATAS. SWEETPOTATO: Monilochaetes infuscans, scurf, was present in most plantings of sweetpotatoes in New Mexico. Market grade was reduced and some fields were not harvested because of the severity of the disease. (P. J. Leyendecker, Jr. PDR 35: 169)

Internal cork (virus). In Oklahoma and California, see Table 1.

LACTUCA SATIVA. LETTUCE: Bremia lactucae, downy mildew, made a sporadic appearance again in the spring of 1950 in Connecticut, according to Rich and Stoddard. The mildew attacked the young plants in cold frames just prior to the time of setting out. (PDR 34: 240) Downy mildew caused serious loss of plants in isolated propagation frames in the vicinity of Burgaw and St. Helena, North Carolina, in January 1950, according to Frank A. Haasis and D. E. Ellis. They reported the effect of soil fumigation and soil drench treatments with fungicides on incidence of downy mildew in the seed bed. The results of this study re-emphasized the importance of applying fungicidal drenches to lettuce seedlings propagated in the cold frame. (PDR 34: 310)

Big-vein (virus) has become of increasing importance in Connecticut since it was first observed in 1946, according to the report by Saul Rich. It causes the greatest loss in head lettuce grown for the early market. Infected heads do not head until the weather becomes warm, by which time the early, high prices are no longer available. Four materials were tested as soil treatments for the field control of this disease. (PDR 34: 253). Rich and Stoddard reported 8 to 10 percent infection of big-vein in Connecticut lettuce plantings (PDR 34: 240)

LYCOPERSICON ESCULENTUM. TOMATO: The results of tests with fungicides for the control of tomato plant diseases in south Georgia in 1949-50 were summarized by Huey I. Borders. (PDR 35: 98)

Botrytis cinerea, gray mold. In Florida Stoner and Hogan reported that the 1949 fall crops and the 1950 spring crops of tomatoes in the Fort Pierce area suffered considerable damage from infection of gray mold. Fruit losses ranged from 100 percent to a trace. About eight different sprays were used. In no case was any discernible control observed. (PDR 34: 210)

Varietal reaction in the field of 17 tomato varieties to blossom-end rot (non-parasitic), Colletotrichum phomoides, anthracnose, and Verticillium albo-atrum, Verticillium wilt, in 1949 and 1950 was reported by W. T. Schroeder. (PDR 35: 160)

Phytophthora infestans, late blight. P. A. Young reported the first epidemic of tomato late blight in central east Texas in 1950. Cool rainy weather from May 11 to June 11, with over 8 inches of rain in 15 days and temperatures of 15° to 25° C. about half of the time facilitated development of the disease. Late blight appeared on fall tomatoes also this year, for the first time in East Texas. (PDR 34: 338). Data on distribution and severity of tomato late blight in Ohio was summarized by J. D. Wilson. Answers to questions concerning the percentage of total acreage treated indicated that approximately 62 percent was sprayed or dusted one or more times. Approximately 60 percent of the acreage showed the disease. Late blight was so severe in some fields that it would not be reasonable to expect that complete control could have been obtained by any means. However, reports indicated that the disease was much less severe in treated fields. (PDR 35: 52). In Louisiana, according to W. J. Martin infected fresh-market tomato fruits may be a source of late blight inoculum. (PDR 34: 236). In the final summary (PDR Suppl. 197: 559) of the plant disease warning service in 1950, Miller and O'Brien reported that late blight of tomato was of economic importance. The severity and loss equalled or possibly exceeded that suffered from the destructive 1946 outbreak. Estimated percentage reduction in yield of late blight infected acreages varied from a trace to 95. Infection sources included airborne spores, potato dump piles, southern-grown plants, and volunteer potato plants. Noteworthy was the spread of the disease westward into Arkansas, Missouri, Iowa and Nebraska. Weather conditions in 1950 were extremely favorable for the development of late blight. Fungicides

used as sprays and dusts were listed, also the formulae, results obtained, and the effectiveness of the control measures.

Sclerotinia sclerotiorum. Roy Bardin reported an unusual occurrence of ascospore infection of tomatoes by S. sclerotiorum in a field of about 20 acres grown for seed production in the Salinas area of Monterey County, California, early in October 1950. (PDR 35: 246)

Stemphylium solani, gray leaf spot. Results of fungicide tests for the control of this leaf spot of tomato in Florida were reported by Robert A. Conover. All fungicides providing zinc ethylene bisdithiocarbamate, with the exception of Parzate, were outstanding in gray leaf spot control. (PDR 34: 182)

Gray wall or vascular browning was reported by L. P. Nichols and Edwin E. Honey in commercial tomato fields and greenhouse plantings and in a few home gardens in Pennsylvania during the summer of 1950. Although gray wall has caused loss in previous years in Pennsylvania, growers are becoming increasingly alarmed about the trouble. During the season of 1950, 33 percent of the crop in one greenhouse and up to 25 percent of the crop in some commercial fields were affected. (PDR 35: 55). Internal or "vascular" browning, a new trouble affecting tomatoes, was observed on commercial truck farms in Virginia for the second year, according to S. B. Fenne. (PDR 34: 352). Evidence from Florida investigations indicated that gray wall or internal browning disease of tomatoes, as it occurs in Florida, is due to physiological factors, according to Warren N. Stoner and William D. Hogan. (PDR 34: 379)

Spotted wilt (virus). In California experiments reported by Michelbacher and others showed that field dusting of tomatoes with DDT controlled thrips very well, but did not result in a proportional reduction of spotted wilt carried by this insect. (PDR 34: 307)

Russet mite. Recurrence of russet mite damage to tomatoes in Texas has been reported by P. A. Young and others. It was recommended that all of the tomato plants in a field be killed to destroy the russet mites and other pests as soon as possible after fruit picking had been completed. This method was very effective in Cherokee County in preventing damage from russet mites in 1950. (PDR 35: 54)

PHASEOLUS VULGARIS. BEAN: Axel L. Andersen reported on incidence of bean diseases in Michigan. Generally infected seed from the 1949 crop, and favorable conditions for spread in 1950, resulted in heavy direct and indirect losses from bacterial blights (Xanthomonas phaseoli and X. phaseoli var. fuscans) and anthracnose (Colletotrichum lindemuthianum). (PDR 35: 89)

PISUM SATIVUM. PEA: E. M. Stoddard and G. A. Zentmyer reported results of Connecticut experiments on the control of damping-off (Pythium spp.) with 8-quinolinol. Trials have been conducted with spinach, peas, eggplant, lettuce, petunias, snapdragons, celery, tomatoes, and elms. (PDR 34: 236)

Uromyces fabae, rust, was observed in July in a home garden on the Oregon coast, according to Frank P. McWhorter. This seemed to be the first report of this rust in Oregon since 1934. The plants in the garden were largely defoliated by the rust, indicating that should the disease become established in commercial peas it would prove extremely destructive. (PDR 34: 269)

Pea enation mosaic virus. In New York, in seed increase fields, W. T. Schroeder analyzed the reaction of 218 varieties and strains of peas to an unusually heavy development of pea enation mosaic virus. Only one line had any healthy appearing plants at the end of the season. (PDR 35: 156)

RHEUM RHAPONTICUM. RHUBARB: Charles Chupp reported that Ascochyta leaf spot of rhubarb developed earlier this year than his records showed for any previous season. Not only were the leaves affected but the disease was serious enough to spot the stems. This is the same fungus which had formerly been named Phyllosticta straminella. (PDR 34: 240)

SOLANUM TUBEROSUM. POTATO: Ditylenchus destructor, potato rot nematode. H. J. Conkle and C. H. Rothe summarized results of the potato rot nematode surveys in 1949 and 1950. These surveys offered reasonable assurance as to the distribution of this pest in the Western States. None was found in two years' survey in California, Colorado, Montana, Nevada, Oregon, or Utah; nor was any found in the 1950 survey in Nebraska or Wyoming. In Idaho, only a few additional infested fields were found, which were close to previously known infestations. (PDR 35: 3)

Heterodera rostochiensis, golden nematode. Feldmesser and Fassuliotis reported on the reaction of the golden nematode of potato to controlled temperatures and to attempted control

measures. (Jour. Wash. Acad. Sci. 40: 355). J. N. Sasser and others reported results of studies on the control of the golden nematode of potatoes with Systox spray (E-1059), an organic phosphate insecticide. (PDR 35: 152)

According to Donald Folsom and Reiner Bonde the skin-spot disease (Oospora pustulans) was more common than usual this spring (1950) on potatoes stored in Maine. (PDR 34: 209)

Phytophthora infestans, late blight. J. R. Wallin, in the north-central region, reported that during the 1950 growing season, eight hygrothermographs were distributed among five potato fields, one tomato field and two inoculated plots. The results indicated that hygrothermographs placed in the field yielded rather accurate information on the temperature-humidity conditions associated with the cyclic development of P. infestans in the field and that they can be successfully used as a basis for forecasting late-blight development. (Phytopath. 41: 38). Harold T. Cook and J. M. Lutz reported data indicating that late blight tuber rot is not likely to be an important storage trouble when blight occurring early is followed by dry weather during the latter part of the growing season and at harvest time. (PDR 34: 15). In the final summary of the plant disease warning service in 1950 (PDR Suppl. 197) Miller and O'Brien pointed out that late blight did not cause as much economic loss on potato as it did on tomato, although it was prevalent in many areas (map p. 564). It was distributed throughout the Atlantic Coast seaboard States and four provinces in Canada. Sources of inoculum included diseased seed potatoes, cull piles, wind-blown spores from more southerly regions, and infected southern-grown tomato plants. Estimated reduction in yield, for the most part did not exceed 10 percent, except in Pennsylvania where reduction in yield amounted to 100 percent in affected fields but averaged 8 percent for the State. Cool-wet weather for extended periods of time during the growing period was ideal for late blight. Fungicides used for potato late blight control included Dithane D-14, nabam and many others (page 568).

Streptomyces scabies, scab. Busch and Gilpatrick described a technique for obtaining developing potato tubers in a relatively short time for inoculation with S. scabies. (PDR 34: 256)

Curly top (virus). R. D. Watson and T. E. Randall reported reaction of several wild seedling potatoes to the curly top virus. (PDR 35: 231)

Rugose mosaic (virus) was reported by M. C. Richards on potato test plots in New Hampshire. On July 1, 1950, 60 percent of the Mohawk and 95 percent of the B 76-43 plants showed infection. A trace only was found in other varieties. (PDR 34: 322)

SOLANUM MELONGENA. EGGPLANT: Verticillium albo-atrum, wilt, has been found in muck and sand land plantings of eggplants in Palm Beach and Martin Counties, Florida, according to Stoner and Hogan. (PDR 34: 213)

TARAXACUM OFFICINALE. DANDELION: D. M. McLean reported Sclerotinia sclerotiorum stem rot, and Ramularia taraxaci, leaf spot, on dandelion grown for seed in Skagit County, Washington. The prevalence of the disease on dandelion only adds to the hosts perpetuating the organism in this concentrated seed-producing area. (PDR 35: 162)

DIVISION OF MYCOLOGY AND DISEASE SURVEY

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 203

PLANT PATHOLOGICAL INVESTIGATION
IN THE UNITED STATES
IV

Supplement 203 June 15, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

PLANT PATHOLOGICAL INVESTIGATION IN THE UNITED STATES IV

PLANT DISEASE RESEARCH AND EXTENSION IN IOWA

W. F. Buchholtz and J. R. Wallin

Plant Disease Reporter
Supplement 203 June 15, 1951

PLANT DISEASE RESEARCH AND EXTENSION IN IOWA

W. F. Buchholtz and J. R. Wallin*

Development and growth of plant disease research and extension in Iowa has been concurrent with the growth and development of the Iowa State College. In keeping with the increasing service of that institution to the agriculture of Iowa and the Nation, the activities of its plant pathologists have been directed toward that objective of service.

The research program has been developed and organized to cope most effectively with disease problems as they have arisen. As a particular plant disease research program was organized and developed, it became known and designated by the staff as the "crown rust program", "corn disease program", "seed treatment program", "watermelon wilt program", "root necrosis program", etc. Certain aspects of such major research programs are listed and briefly discussed in this presentation.

MAJOR FIELDS OF RESEARCH OF LONG DURATION

A record of plant disease research in Iowa should perhaps begin with recognition of early observations and descriptive contributions by L. H. Pammel. As a diligent student of the indigenous and economic plants of Iowa, he was very much aware of their maladies, and took time to record and describe some of them and to teach others to do likewise. Two of his noteworthy contributions were the description of Xanthomonas (Bacillus) campestris as the cause of black rot of rutabaga in 1895 (67) and the description of Helminthosporium sativum (with King and Bakke) as the cause of barley blight (68) in 1910.

Under Pammel in the Department of Botany, I. E. Melhus began his service to Iowa State College as plant pathologist in 1916. His appointment marked the beginning of an intensive effort in plant disease research and teaching which, as the staff and the College grew, resulted in the major research and extension programs which are summarized in this discussion.

CROWN RUST OF OATS: -- Oats has come to be Iowa's major small grain crop, and Puccinia coronata has been the oat crop's most damaging and persistent pathogen. The Iowa Agricultural Experiment Station has carried forward a crown rust research program continuously for many years. I. E. Melhus inaugurated the project in 1914 and has maintained an effective constant interest in it. Such able investigators as J. H. Parker, L. W. Durrell, Florence Willey, L. D. Leach, S. M. Dietz and H. C. Murphy have been associated with or in charge of the project. The U. S. Department of Agriculture has cooperated with the Iowa Station ever since the initiation of this work. As a Federal employee, S. M. Dietz was in charge during the ten-year period 1917 to 1927. In 1928, H. C. Murphy assumed responsibility for the project and since then has carried it forward, along with some consideration of other oat pathogens, notably Helminthosporium victoriae. Breeding for crown rust-resistance has been in cooperation with plant breeders at the Iowa Station.

Early accomplishments were a clear definition of the crown rust problem (50), determination of the Rhamnus host range of P. coronata (49), recognition of the phenomenon of specialization in P. coronata (49, 60), and the incorporation of the Green Russian type of resistance into such strains as D67 and D69 (11), which served as useful parents in the later resistance program.

A landmark of progress was the discovery, in 1929 (63) and 1931 (96), of the lines, Victoria and Bond, respectively, whose crown rust- and smut-resistance were combined with the stem rust-resistance of Richland (97), Iogold, and D69 (64). The Victoria-Richland varieties, Boone, Tama and Control, were made available to Iowa farmers in 1941, and by 1945 constituted an estimated 98 percent of the Iowa oat acreage. Clinton, the result of a D69-Bond cross (61), was first grown in 1945, and by 1950 Clinton and other derivatives of this cross constituted about 95 percent of the Iowa acreage.

According to a recent conservative estimate, since 1941 Iowa farmers have produced 255,500,000 bushels more oats by growing these crown rust-resistant varieties.

Change in the race constitution of P. coronata may force a change from the varieties carrying the resistance of Bond. Varieties are in the making carrying the resistance of such varieties as Landhafer, Santa Fe, Trispernia, and Ukraine to the now prevalent crown rust races that parasitize Clinton and other Bond derivatives. The Rhamnus host range of these races is in

* C. S. Reddy, G. Semeniuk, H. C. Murphy, J. C. Gilman, W. J. Hooker, and J. R. Crall provided very useful information and suggestions for this summary.

process of determination. Through the years, the prevalent races of P. coronata have been determined and recorded.

A serious impediment to the crown rust-resistance program was the sudden and destructive outbreak of Helminthosporium victoriae which, during the period 1945, 1946, and 1947, caused as severe damage to Victoria derivatives as had earlier epiphytotics of P. coronata. This unexpected threat was met by definitely ascribing the damage to H. victoriae (42), by promptly ascertaining that the only hosts were Victoria and its derivatives (62), by establishing that susceptibility of these varieties to H. victoriae was irrevocably "linked" to resistance to P. coronata (38), and by revising the crown rust-resistance program to avoid further use of Victoria and its derivatives.

CORN DISEASES: -- Intensive corn disease research at the Iowa Station was begun by I. E. Melhus in 1920, following the early observations made by Pammel and his associates (69, 70). Early associates of Melhus on the corn work were L. W. Durrell, F. H. van Haltern, C. S. Reddy, D. E. Bliss, G. A. Platz, A. H. Eddins, W. P. Raleigh, and Mary F. Howe. In 1937 a full time research position was established and held for one year by G. N. Davis; since 1938 George Semeniuk has held this position and has had responsibility for a major portion of corn disease research, although the importance of the crop in Iowa has been the basis for the continued efforts of Melhus, Reddy and their students. Corn disease research at the Iowa Station has been supported entirely with State and gift funds. The disease resistance aspects have been studied cooperatively with agronomists.

Ear, stalk and root disease investigations have been stressed continuously over the years. The ear rots caused by Diplodia zeae, Gibberella zeae, and Nigrospora oryzae received first attention because of their prevalence and their importance to the selection of ears for seed, (14, 15, 16, 69, 70, 76). Their deleterious effects on the vitality of the seed and on the health of the seedling (14, 77, 79) led to recommendations for their detection and control by seed disinfection (14 54, 76). Cob acidity greater than pH 5.2 (80), characteristic of matured ears (94, 95), and the process of seed germination (80) were found to confer resistance to Nigrospora orzyae. This fungus was found to develop and persist profusely on the rudimentary ears in the lower leaf axils (95).

Stalk rot disease investigations have dealt primarily with Diplodia zeae. Durrell, G. L. McNew and Semeniuk have participated in various phases of the Diplodia problem. Their results showed that the pathogen: (a) infected the plant at the nodes following a period of development on dead pollen lodged between the leaf sheaths and stalks (13); (b) was destructive to seedlings when introduced into the soil (18, 87, 89), and that by this method susceptible single crosses were differentiated (88); and (c) invaded the mesocotyl and crown tissues of young plants but remained inactive until the plants approached maturity (40).

Investigation of root rots, which are of major significance to Iowa corn production, have resulted so far in description of the symptoms produced by Pythium graminicola, Helminthosporium sativum, Gibberella zeae, Rhizoctonia solani and other pathogens (25), and evaluation of seedling resistance of some lines to P. graminicola (25, 93).

Smut has been studied at intervals. Factors influencing spore germination (74), and the pathogenicity of Ustilago zeae (= U. maydis) (17, 73, 119) were the subject of early inquiries. These were followed by development of the fish-oil soap (10) and similar (119) techniques for inducing the passage of a sporidial suspension to the growing point of the young plant via the spiral whorl. The concept of the systemic infection of the corn plant by the smut fungus (10) resulted from use of these techniques and from the observation that rudimentary galls were common at all the nodes of infected plants. New methods of inoculation, by hypodermic injection of sporidial suspensions into the unopened coleoptiles (98) or at the first node of the stem on germinating seed, were devised in the search for a more rapid process of evaluating resistance among lines of corn. The discovery that seedling infection may follow the application of sporidia to germinating seed (111) provided a simple method for inducing seedling infection and demonstrated another avenue through which infection can occur.

Other corn disease contributions include an account of the purple leaf sheath spot of corn (13); experimental demonstration of seedling infection by oospores of Sclerospora graminicola taken from Setaria, and a record of occurrence of this pathogen on corn in Iowa (19, 55, 56); a description of the Holcus leaf spot bacterium (Pseudomonas holci) and the symptoms induced by it (34); and proof that seedlings as well as maturing stalks can be attacked by Sclerotium bataticola (90).

When shelled corn was under loan and seal in steel bins throughout Iowa, the Iowa Station, in cooperation with the Commodity Credit Corporation, made extensive studies of stored corn

deterioration; including those of Semeniuk and associates (66, 91, 92) on the role of molds in stored corn spoilage. The practical result of this research was the realization that moisture contents must be lower than 13.5 percent to permit continuous storage of shelled corn.

One of the major objectives of the Iowa State College Guatemala Tropical Research Center has been to collect disease-resistant corn germ plasm. A large number of collections have been evaluated for resistance in the seedling stage to Pythium graminicola (93), and some for resistance to Helminthosporium turcicum (101). Degrees of H. turcicum and rust attack have been noted on many lines in Guatemala (48).

Because, up to now, no single plant pathogen has been a limiting factor in corn production in Iowa, comparable to Puccinia coronata on oats, it has not been necessary or readily possible to apply the results of corn disease research to crop production so intensively. The potential value of the corn disease knowledge contributed from Iowa and other States and the U. S. Department of Agriculture, however, is available to all those interested in corn. Many corn breeders are aware that corn is "good" partly because it is disease-resistant. Full exploitation of this approach to "corn improvement" remains for the future, but the overall value of what has been accomplished on this important and widely grown crop has already been very substantial.

SEED TREATMENT: -- Plant Pathologists at the Iowa Station have been closely identified with the development of seed treatment as an agricultural practice. Melhus, J. C. Gilman and J. B. Kendrick, in 1920, determined the efficacy of hot formaldehyde in disinfecting potato seed tubers (51). Associates J. J. Wilson, W. P. Raleigh and C. S. Reddy developed Merko and Sterocide as corn seed disinfectants in 1928 and 1930, respectively. With the increased activity of large chemical companies in producing new basic fungicidal compounds, the interest of the Iowa Station turned to evaluation of compounds and formulations and to defining their activity and usefulness under Iowa conditions. Such efforts were stimulated in part by studies of effectiveness of corn seed treatment at different planting rates (81) and at different planting dates, of flax seed treatment at different planting dates (7, 83) and of sugar beet seed treatment (85).

These studies contributed to a growing realization that a fungicide coating can serve as a protectant of the germinating seed against soil-borne pathogens, and that its effectiveness is conditioned by soil temperature and moisture, in a manner and degree depending on the crop. Corn seed, for instance, has been found to be most benefited by seed treatment when subjected to "cold and wet" soil conditions soon after planting (86); likewise flax (83). Field experiments showed that treated seed of corn and flax could be planted with safety somewhat earlier than untreated seed (82, 83), and that seed treatment of relatively disease-free oats seed, by protecting such seed from soil-borne pathogens, resulted in increased yields. During the past ten years, even with relatively disease-free seed, corn and oat yields have been increased an average of more than three bushels per acre by recommended fungicide seed treatments (84).

An unusual by-product of the knowledge that at low temperatures corn seed is attacked by soil-borne organisms has been the "cold-test" for seed corn (86). The fact that high quality corn seed, in addition to being viable, is capable of surviving for a short time in Pythium-infested soil at low temperatures, in contrast to the very rapid deterioration of low quality seed under such circumstances, is now so well recognized that the "cold-test" has become a standard seed laboratory germination and seed treatment evaluation technique. A similar test, in which the seed is flooded at room temperature, is now being developed.

Most of this knowledge has resulted from contributions by Reddy and his associates.

Recently seed treatment research has been directed toward evaluating the role of fungicides in establishing stands of soybeans and small seeded forage legumes and grasses. A beginning has been made in determining their effect on legume root nodulation in Iowa soils.

WATERMELON WILT: -- Devastation of the watermelon industry in southeastern Iowa by the wilt pathogen led to inauguration of the watermelon wilt project in 1925. Under the guidance of Melhus there was first a definition of the problem and a study of the mode of parasitism by Fusarium niveum (75, 120). In 1930 the resistant varieties Iowa Belle, Iowa King, and Pride of Muscatine were available. All were developed by mass selection and selfing within susceptible varieties grown on infested soil. By 1937, Stone Mountain #5 and Kleckley #6 had been developed, both from further exploitation of the same technique. By the back-crossing method, it was possible to combine anthracnose- and wilt-resistance in several lines. One of these is the variety, Black Kleckley. Continued efforts resulted in the production of Kleckley Hybrid (1941) and Dixie Hybrid (1941), the latter an unusually high quality wilt-resistant melon, the first such available to Iowa growers (84).

After twenty years, many southeastern Iowa watermelon growers have turned to other crops. The way to recovery of the industry has been opened, however. Furthermore, wilt- and anthracnose-resistant germ plasm developed in Iowa has been utilized in disease-resistance programs in a number of other States.

NURSERY CROP DISEASES: -- In Shenandoah and Hamburg, in southwestern Iowa, are the headquarters for three large nurseries, one of which is recognized as this country's largest wholesale producer of nursery stock. Several nursery disease problems have been investigated, notably crowngall, cherry leaf spot and mildew, cedar apple rust, damping-off of conifers and other seedlings, and Phomopsis blight of cedar. Interest and activity in solving nursery disease problems has been of 30 years' duration, with some attention being given by Melhus, J. H. Muncie, R. F. Suit, D. E. Bliss, and G. L. McNew. Among the practical results are a clearer definition of the crowngall and callus knot problem (43, 52, 57, 59), apple grafting techniques that limit development of callus knot and crowngall at the graft union (53), satisfactory cherry fungicide sprays for leaf spot and mildew control (41), a record of the seasonal development and pathogenicity of Gymnosporangium in Iowa (3), and a seed-bed treatment for conifer damping-off control. Research contact with the nursery industry has permitted making many suggestions for disease control by practices developed by research other than in Iowa.

SUGAR BEET DISEASE: -- Diseases have been a major factor in the discouragement of sugar beet production in northern Iowa. In an attempt to meet this problem, the Cercospora leaf spot and Aphanomyces root rot diseases were intensively investigated on the Northern Iowa Experimental Association farm at Kanawha, beginning in 1930. E. F. Vestal, F. G. Bell, C. M. Nagel, C. S. Reddy and W. F. Buchholtz have been full or part time workers in this field. Wider spacing with cross-blocking to provide aeration for leaf spot control (103, 65), seed treatment to prevent seed decay and seedling damping-off (35), crop rotation to avoid heavy leaf spot and root rot losses (6), and a means of indexing soil to predict losses from Aphanomyces root rot in a field to be planted to beets (20), are among the practical results of this research.

High returns from unrestricted acreages of corn, soybeans, and crown rust-resistant oats during the 1940's have helped discourage a return to sugar beet production, and varieties somewhat resistant to Cercospora beticola and Aphanomyces cochlioides may be needed to help insure stability, if or when conditions are again favorable to sugar beet production in northern Iowa. The supplementary control practices developed and demonstrated by plant pathologists at the Iowa Station will be valuable.

ROOT NECROSIS: -- For many years an active root necrosis research program has been conducted at the Iowa Station. Early experiences of Melhus, Reddy and others with corn seedling blight and Diplodia stalk rot were a beginning. Work by Buchholtz on sugar beet root rot and sugar beet and legume damping-off (4, 5, 6) emphasized the role of soil-borne phycomycetous pathogens of those crops. Reddy's and his associates' discoveries led to the realization that corn seed rotting in cold soil was by Pythiaceous fungi, (82, 86); Wen-Chun Ho, C. H. Meredith and Melhus demonstrated conclusively that Pythium graminicola was a major pathogen of corn and barley roots (25, 26); A. W. Welch did likewise for P. debaryanum and P. irregulare on oats (117) -- not P. graminicola, which has been repeatedly substantiated by later work; S. G. Younkin's work indicated that "wilt" of very young watermelon seedlings, which was common even with varieties resistant to Fusarium niveum, was probably damping-off by P. irregulare (122). Subsequent work, some of it yet unpublished, has shown that there may be a seasonal variation in the Pythium "content" of infested soil (39); that P. graminicola is most abundant in the upper 12 inches of soil (37); that moisture deficiency accentuates the effect of Pythium injury to barley (39); that with time there is a progressive decrease in occurrence of P. debaryanum and an increase of P. graminicola on barley seedlings and roots, which may be in response to seedling growth as well as to season (100); and that there are measurable differences in susceptibility to P. graminicola between varieties of corn (25, 93), barley (23, 26) and bromegrass (24).

The role of phycomycetous and other root pathogens in crop and variety choice and all aspects of crop production in Iowa is well indicated but not yet fully defined. There seems little doubt that P. graminicola was in large part responsible for failure of the barley crop in Iowa during 1942-1944, and that stability of barley production in Iowa will depend in part on a variety with considerable resistance to this pathogen. The root necrosis research completed and under way enabled realization of that fact and paved the way for the developing of a resistant variety (23).

Root necrosis research is going forward at the Iowa Station.

BACTERIAL PLANT PATHOGENS: -- A broad and flexible program of bacterial disease research has been fostered, not only to permit effective and prompt investigation of timely problems, but also to encourage contributions to an understanding of the basic concepts concerning the bacterial plant pathogens as a group.

Research on the bacterial plant pathogens began with L. H. Pammel's work on Xanthomonas campestris in 1895 (67). From 1919 to 1937, crowngall and hairy root of nursery stock were studied. Investigations on these diseases were stimulated by alarmed Iowa nurserymen who were losing thousands of trees, supposedly because of crowngall. They supported, in part, the crowngall research program in Iowa and Wisconsin through the Crop Protection Institute. In Iowa, I. E. Melhus, J. H. Muncie, Wm. T. H. Ho, M. K. Patel, R. F. Suit and G. C. Kent were associated with either or both crowngall and hairy root investigations.

Briefly, some of the results of these studies were: an improved method of isolating the crowngall bacterium (71); discovery that a high percentage of galls on apple grafts were callus knot rather than crowngall (59); and that callus knot could be reduced by carefully fitted wedge grafts (53); confirmation of Riker and Keitt's earlier report that hairy root was caused by a distinct species of bacteria, Agrobacterium rhizogenes (99); isolation and culture of a phage specific for A. tumefaciens (35, 58).

The bacterial diseases of cereal crops were investigated by J. B. Kendrick, C. H. Kingsolver, C. S. Reddy and J. R. Wallin. The contributions resulting from their work include: the description and designation of Pseudomonas holci as the cause of leaf spot on corn and sorghum, (34); the determination that Clinton and other oat selections were resistant to the halo blight bacterium, Pseudomonas coronafaciens (36); the description and naming of Xanthomonas translucens var. phleipratensis on timothy (114); the description, naming and determination of the host range of X. translucens var. cerealis found on bromegrass (109); and the demonstration that X. translucens on infested barley, bromegrass and wheat seed will infect the developing seedling (110).

T. F. Yu, S. T. Chao, and W. J. Hooker have studied the bacterial wilt of cucumbers, the bacterial pustule and blight of soybeans, and potato scab, respectively. Two noteworthy contributions were: elucidation of the process of wilting in cucumbers induced by Erwinia tracheiphila and determination of varietal reaction to the pathogen (121); and demonstration of overwintering of the bacterial pustule and blight organisms, Xanthomonas phaseoli var. sojense and Pseudomonas glycine respectively, in soil, soybean seeds and debris (8). The potato scab work will be discussed in a subsequent portion.

Investigations relating to the differentiation and classification of certain phytopathogenic bacteria have been fostered by Melhus. M. K. Patel, G. C. Kent, E. L. Waldee, and V. P. Bhide participated in the work. Their results included: determination that pathogenicity was a necessary criterion for identification of Agrobacterium tumefaciens (72); the proposal of a new family and genus, Erwiniaceae and Pectobacterium, respectively, to include the emended genus Erwinia and related genera (108); and the separation of nine wilt-producing bacteria belonging to four genera into two groups based on their enzymatic activity, nitrogen utilization, and growth temperature range (2).

SPECIAL PHYTOPATHOLOGICAL INVESTIGATIONS: -- From time to time related physiological problems have been investigated. For example, Hoyman (32a) characterized the emetic principle present in scabby barley.

NEW FIELDS OF RESEARCH GIVEN MAJOR EMPHASIS SINCE 1940

Such trends and events as the fabulous increase in soybean acreage, growing interest in improved forage legumes and grasses, and war-time emergency forecasting of potato late blight occurrence led to recent development of research programs in these fields. In the meantime, advances in knowledge of stone fruit viruses made it imperative that Iowa nurserymen begin producing virus-free stocks as soon as possible, the potato scab problem became so acute that an intensive research program was urgent, and Iowa oak trees were suffering from the wilt disease. Research programs dealing with these three problems have also been developed at the Iowa Station since 1940.

POTATO SCAB: -- Although the potato scab problem had been under investigation for some time, particularly in aspects relating to fungicidal control through seed piece treatment, it was not until 1940 that an intensive effort was begun to develop control measures for Iowa's most serious potato disease. After inauguration of the project by Melhus, with the help of G. C. Kent, W. J. Hooker assumed responsibility for the work in 1944. Careful observations revealed that in

Iowa muck soils, Streptomyces scabies could and did parasitize roots and soil-covered stems of potato (32); also that under laboratory conditions, seedling roots of a wide range of plants were susceptible to S. scabies (27). Attempts are being made to induce scab development under controlled conditions in the greenhouse, to lay the basis for testing seedling resistance to S. scabies. A means of observing growth of potato tubers (28) has made possible the direct inoculation of tubers with pure cultures. Thus the progressive development of scab infections has been observed (31).

The effectiveness of control by sulfur and other amendments to Iowa muck soil has been determined (30) and the toxic effect of sulfur has been shown to reduce Streptomyces spp. populations before the soil reaction has changed markedly (104).

As a result of Hooker's interest, other vegetable disease research has been undertaken, notably potato and tomato early and late blight control by fungicidal sprays (29), onion smut control by large dosages of seed protectant chemicals, and sweetpotato pox control by soil amendment with sulfur. In recent years, through cooperation with C. E. Peterson of the U. S. Department of Agriculture in the Department of Horticulture, greenhouse tests have been made for resistance in parents and seedling progenies of potatoes to Phytophthora infestans and viruses. Tomato introductions in the hands of the Plant Introduction Station have been similarly tested for resistance to the Septoria and Alternaria blight pathogens.

SOYBEAN AND FORAGE CROP DISEASES: -- The increasing importance of the soybean in Iowa agriculture, plus the post-war interest in "seeding down" land heavily cropped during war-time, have focused attention on diseases of legumes and forage grasses. Beginning in 1946, A. W. Welch started the intensive program in this field, under projects supported in part by the U. S. Department of Agriculture. Since 1948, J. M. Crall has been carrying forward this work, particularly with legumes. J. C. Gilman and Lois H. Tiffany have helped with some parts. Efforts and contributions have included elucidation of the pod and stem blight and anthracnose complexes on soybeans (102, 118), and of the anthracnoses of red clover (22); also the discovery of a wilt of red clover caused by Fusarium oxysporum (9). Clarification of the role of seed treatment of legumes has been discussed under "seed treatment".

Forage grass disease research was begun by Welch. One of his contributions was the demonstration that "northern" bromegrass was very susceptible to Pythium graminicola (24); northern bromegrass is less likely to develop good stands in Iowa than the "southern" form, which is not so susceptible.

LATE BLIGHT FORECASTING: -- Two factors stimulated the initiation of potato late blight forecasting in Iowa, namely: the prevalence and destructiveness of late blight in the Upper Mississippi Valley region during 1942, and the war-time crop protection emergency.

The destructive potato late blight epiphytotic in 1942 prompted Melhus (46) to propose a study of the temperature-rainfall conditions associated with past Iowa late blight epiphytotics. His studies revealed that for the establishment and spread of the pathogen, relatively low temperature and high rainfall must prevail throughout June and July. Melhus' findings served as a background for the regional late blight forecasting effort which was to follow.

During the national emergency of World War II, plant pathologists were greatly concerned about plant disease control as a means of increasing the nation's food supply. As chairman of the Late Blight Subcommittee of the Upper Mississippi Valley Plant Pathologists' War Service Committee, Melhus led a coordinated effort to report the occurrence and development of late blight in this region. Furthermore, upon receipt of information regarding temperature-rainfall and blight occurrence, forecasts for the region were formulated. Throughout the war emergency period several relatively accurate forecasts were issued and much information about late blight and other diseases was exchanged (47).

Buchholtz resumed a plant disease information exchange in 1946 as the result of an extremely costly tomato late blight epiphytotic in Iowa and the Upper Mississippi Valley. He functioned as an information clearing agent not only for the Iowa extension people, but also for a voluntary group of extension plant pathologists in several States. He continued this work until 1948, at which time the voluntary reporting in the region was undertaken as a part of the U. S. Department of Agriculture regional crop plant disease forecasting project.

In 1948, the U. S. Department of Agriculture Division of Mycology and Plant Disease Survey initiated its crop plant disease forecasting project in the North Central Region. Iowa State College was designated as regional headquarters, and J. R. Wallin was appointed to conduct research basic to tomato and potato late blight forecasting in the North Central States. Within a year after initiation of the project, P. E. Waggoner was employed to assist with the work in Iowa.

The following contributions have resulted from the regional research program:

(a) From a focus of infection, Phytophthora infestans was observed to spread slowly but persistently under conditions that appeared unfavorable (106, 116).
(b) In tomato and potato plots only minor differences were noted between air temperature and humidity at the five-foot and the one-foot levels (105, 116).
(c) Within five Fahrenheit degrees, air temperatures in the instrument shelter were found to represent the temperature of the air surrounding tomato and potato plants, the temperatures of shaded leaves in the daytime and all plant parts at night. However, sunlit plant parts were found to be more than 5° F. warmer than the surrounding air temperature (107).
(d) Fluctuations in amount of late blight were shown to depend upon the frequency of occurrence of favorable temperature and humidity (112, 115).
(e) Isolates of P. infestans have been characterized by their pathogenicity on potato and tomato in the field (113), greenhouse, and laboratory.

These and further efforts supported by this project promise to provide data that will enable the anticipation of late blight epiphytotics with sufficient accuracy to permit growers of the region to intensify or relax fungicidal spray programs on potatoes and tomatoes, with a prospective saving either of the crop or of labor and fungicide cost.

STONE FRUIT VIRUS DISEASES: -- In keeping with the stone fruit industry's demand for virus-free planting stocks from Iowa's wholesale producers (45), there was inaugurated in 1947 a project designed to facilitate production of such virus-free stocks of cherries, peaches and ultimately plums and perhaps all Prunus species and varieties. Thus far, indexed virus-free clones of sour cherries and some sweet cherry varieties have been collected and established. The Mount Arbor orchards of Prunus mahaleb have been indexed and known infected trees discarded.

As a means of determining necessary isolation for virus-free nursery stocks of cherries, samples of Prunus species and varieties cultivated in Iowa nurseries have been virus-indexed at intervals. Some spread was indicated within cherry blocks containing infected trees (21).

One Iowa nursery has been selling cherry trees propagated from indexed scion trees. The three large producers are now in production, although the lack of virus-free understocks remains as an impediment in the program. Understock production is largely in the Pacific Coast States of Washington, Oregon and California.

This work has been under the supervision of W. F. Buchholtz, with H. C. Fink and O. F. Hobart Jr. as student contributors.

OAK WILT: -- Investigation of the oak wilt disease was begun in 1943, at which time the Iowa State Conservation Commission provided resources to determine the cause of oak tree decline in Iowa's State parks. S. M. Dietz, J. W. Barrett, R. A. Young, G. L. McNew, Paul Hoffman, and W. H. Bragonier have at various times been associated with this project. Contributions to date include confirmation that Chalara quercina is the cause of oak wilt in Iowa, and determination of its prevalence in Iowa oaks, as well as its host range under greenhouse conditions (12).

EXTENSION PROGRAM

The Iowa Extension program in plant pathology has been modest in personnel and resources expended, but its activities have influenced agricultural practice in Iowa and the Midwest.

PROMOTION OF ADOPTION OF DISEASE-RESISTANT VARIETIES AND DISEASE-FREE SEED STOCKS: -- The Iowa State College Agricultural Extension Service has long been active in promoting the use of disease-free seed stocks and disease-resistant crop varieties. Adoption of yellows-resistant cabbage and rust-resistant Washington asparagus in southeastern Iowa was promoted by Melhus and his associates before plant disease teaching, as such, was a well established activity of the Extension Service.

Discoveries by workers elsewhere that viruses cause "running-out" of potatoes, and the establishment of production of virus-free seed stocks in northern and western States, formed the basis for teaching growers and gardeners the value of "certified" (disease-free) seed potatoes.

Early success with such projects soon led to the establishment of a position for a plant pathologist in the Extension Service. After a short time of service by R. O. Cromwell, R. H. Porter was appointed to this post in 1921. Except for an interim spent in China, during which time Morrison Burns served, Porter was Extension Plant Pathologist until 1933. Then for a time D. V. Layton served in that capacity. Later came J. H. Standen, E. L. Waldee, and D. M. Coe. A. F. Sherf is the present Extension Specialist in Plant Pathology. From time to time, E. P. Sylvester has aided in the program as the occasion demanded. Since 1936, C. S. Reddy has devoted part time to Extension plant pathology.

Assignment of personnel resulted in expansion and intensification of the plant pathology program in Extension. In addition, the newly acquired phytopathological research farms at Kanawha and Conesville offered unusually effective facilities for field demonstration. One such demonstration at Kanawha showed that wilt-resistant flax varieties would actually produce satisfactory crops for five successive years on the same field, while susceptible varieties failed after the first year. Dissemination of such information played a large role in resumption of flax growing in Iowa, an enterprise which, with war-time prices, reached an all time high of over 350,000 acres in northern Iowa in 1943.

By participating in field demonstrations and dissemination of information, verbal and printed, Extension plant pathologists have helped promote the use of the crown rust-resistant oat varieties available to Iowa farmers since 1940. An additional 110 million bushels of oats produced by Iowa farmers during the years 1941 to 1945 were in part the result of an intensive war-time Extension campaign (1) to promote adoption of the crown rust-resistant varieties responsible for the additional yield.

Through Extension activities at Conesville in southeastern Iowa, availability and performance of wilt-resistant varieties of watermelons have been made known to growers in that area. Nearly all watermelons grown in that area since 1930 have been of such wilt-resistant varieties.

PROMOTION OF FUNGICIDE SEED TREATMENT AS AN AGRICULTURAL PRACTICE, AND THE ADOPTION OF CONVENIENT AND ECONOMICAL METHODS OF SEED TREATMENT APPLICATION: -- The first bulletin recommending seed treatment of corn was puslished by the Iowa State College in 1927 (54). The adoption of seed treatment as an agricultural practice in Iowa has paralleled the striking growth of knowledge in this field and the development of suitable fungicide seed dusts.

The first objective of cereal seed treatment, smut control (with formaldehyde), was sponsored by Iowa State College as early as 1918 (33). The advent of the organic mercury dusts for control of smut and seedling blight caused by seed-borne pathogens was the basis for an extensive series of demonstrations of the economic value of seed treatment and of the revolving barrel and gravity methods of application, by Porter and Layton (78). During the late 1930's, with the development of machines for continuous application and of knowledge that seed treatment could protect the germinating seed from damage by soil-borne pathogens, there was a tremendous increase in custom seed cleaning and treating in farm and city elevators. Practical demonstrations on a commercial basis at Kanawha and Conesville played a large part in promoting both seed treating and seed cleaning, the latter a worthwhile weed control as well as disease control practice. Custom seed cleaning and treating has been an annual demonstration at Kanawha since 1935. At least a million bushels of cereals, corn, and soybeans have been so processed there. In 1950, there were 240 seed cleaning and treating stations in Iowa.

Some form of seed treatment fungicide is applied by hybrid corn processors to practically every bushel of seed corn produced or sold, or both, in Iowa. Original promotion of this procedure was in part by Extension plant pathologists in Iowa and other States. There is no longer a need to induce seed companies to treat corn seed, but they have been constantly advised about latest developments in fungicide chemicals and formulations and methods of application by Iowa Extension plant pathologists.

Although development of wilt-resistant varieties was basic to the resumption of flax production in northern Iowa, seed treatment, by insuring good stands with early plantings, was a large factor in the successful wartime production of large flax acreages in northern Iowa. Burnett and Reddy presented data on the first successful flax seed treatments (7). Demonstrations and reports (84) of experiments at the Northern Iowa Experimental Association farm at Kanawha were the basis for adoption of flax seed treatment as an agricultural practice in Iowa, and possibly for other flax-producing States.

PROMOTION OF SPRAYING FOR FRUIT AND VEGETABLE DISEASE CONTROL: -- Iowa fruit and vegetable growers and gardeners are regularly advised as to new and appropriate

disease control measures. Commercial growers of apples and potatoes practice scab and early and late blight control, respectively, according to recommendations by plant pathologists, made through facilities provided by the Extension Service.

LATE BLIGHT FORECASTING: -- The potato and tomato late blight forecasting initiated in Iowa during the war was followed closely by the Extension Services of Iowa and surrounding States. They used the predictions to stimulate growers to practice more effective spraying or to refrain from spraying and save valuable materials and scarce labor, whichever the local situation warranted. The three years' war-time service has been summarized (47), and a saving of several million bushels of potatoes estimated for the region.

VICTORY GARDEN PROGRAMS: -- Another significant war-time Extension activity by Iowa plant pathologists was participation in the victory garden campaign. Through personal appearances at education meetings and through bulletins, the press and radio, the place of disease control in home gardening and orcharding was made known to victory gardeners throughout the State.

Literature Cited

1. Anonymous. Increase crops through disease control (oat diseases). Iowa State Col. Agr. Ext. Serv. Pamphlet 25. 1942.
2. Bhide, V. P. A comparative study of some wilt-producing phytopathogenic bacteria. Indian Phytopath. 1: 70-91. 1948.
3. Bliss, D. E. The pathogenicity and seasonal development of Gymnosporangium in Iowa. Iowa Agr. Expt. Sta. Res. Bul. 166. 1933.
4. Buchholtz, W. F. Factors influencing the pathogenicity of Pythium debaryanum on sugar beet seedlings. Phytopath. 28: 448-475. 1938.
5. _____. Influence of cultural factors on alfalfa seedlings infection by Pythium debaryanum Hesse. Iowa Agr. Expt. Sta. Res. Bul. 296. 1942.
6. _____. Crop rotation and soil drainage effects on sugar beet tip rot and susceptibility of other crops to Aphanomyces cochlioides. Phytopath. 34: 805-812. 1944.
7. Burnett, L. C., and C. S. Reddy. Seed-treatment and date-of-sowing experiments with six varieties of flax. Phytopath. 21: 985-989. 1931.
8. Chao, S. T. Bacterial blight and bacterial pustule of soybean. Ph.D. Thesis, Iowa State Col. Library. 1946.
9. Crall, J. M. Wilt of red clover seedlings. Phytopath. 41: 7. (Abst.) 1951.
10. Davis, G. N. Some of the factors influencing the infection and pathogenicity of Ustilago zeae (Beckm.) Unger on Zea mays L. Iowa Agr. Expt. Sta. Res. Bul. 199. 1936.
11. Dietz, S. M. Inheritance of resistance in oats to Puccinia graminis avenae. Jour. Agr. Res. 37: 1-23. 1928.
12. _____, and R. A. Young. Oak wilt -- a serious disease in Iowa. Iowa Agr. Expt. Sta. Ext. Serv. Bul. P91. 1948.
13. Durrell, L. W. A preliminary study of the purple leaf sheath spot of corn. Phytopath. 10: 487-495. 1920.
14. _____. Dry rot of corn. Iowa Agr. Expt. Sta. Res. Bul. 77. 1923.
15. _____. Basisporium dry rot of corn. Iowa Agr. Expt. Sta. Res. Bul. 84. 1925.
16. _____, and D. R. Porter. Dry rots of corn and their control. Iowa Agr. Ext. Serv. Bul. 122. 1924.
17. Eddins, A. H. Pathogenicity and cultural behavior of Ustilago zeae (Beckm.) Unger from different localities. Phytopath. 19: 885-916. 1929.
18. Erwin, L. E. Experiments on germination and infection with Diplodia zeae (Schw.) Lév. M.S. Thesis, Iowa State Col. Library. 1930.
19. Evans, M. M., and G. Harrar. Germination of the oospores of Sclerospora graminicola (Sacc.) Schroet. Phytopath. 20: 993-997. 1930.
20. Fink, H. C. Correlation between sugar beet crop losses and greenhouse determinations of soil infestations by Aphanomyces cochlioides. M.S. Thesis, Iowa State Col. Library. 1947.
21. _____. Detection of necrosis ring spot virus and its occurrence and spread in sour cherry nurseries. Ph.D. Thesis, Iowa State Col. Library. 1950.

22. Gilman, J. C., and Lois H. Tiffany. Some species of Colletotrichum on leguminous forage crops. Phytopath. 40: 10. (Abst.) 1950.
23. Haskett, W. C. Relative susceptibility of barley varieties to Pythium graminicola. M.S. Thesis. Iowa State Col. Library. 1951.
24. Hawk, V. B., and Z. W. Welch. Tolerance of varieties of smooth bromegrass, Bromus inermis Leyss., to injury by root rot, Pythium graminicola Subr. Jour. Amer. Soc. Agron. 40: 809-817. 1948.
25. Ho, Wen Chun. Soil-inhabiting fungi attacking the roots of maize. Iowa Agr. Expt. Sta. Res. Bul. 332. 1944.
26. _____, C. H. Meredith, and I. E. Melhus. Pythium graminicola Subr. on barley. Iowa Agr. Expt. Sta. Res. Bul. 287. 1941.
27. Hooker, W. J. Parasitic action of Streptomyces scabies on roots of seedlings. Phytopath. 39: 442-462. 1949.
28. _____. A technique for observing tuber enlargement and scab development in potatoes. Phytopath. 40: 390-391. 1950.
29. _____, W. F. Buchholtz and D. M. Coe. You can whip potato blight. Iowa Farm Science, Vol. 4, No. 12: 188-189. 1950.
30. _____, and G. C. Kent. Sulfur and certain soil amendments for potato scab control in the peat soils of Northern Iowa. Amer. Potato Jour. 27: 343-365. 1950.
31. _____, and O. T. Page. Potato tuber growth and scab infection. Phytopath. 41: 17. (abst.) 1951.
32. _____, J. E. Sass and G. C. Kent. Stem necrosis of potatoes caused by Streptomyces scabies. Phytopath. 40: 464-476. 1950.
32a. Hoyman, Wm. G. Concentration and characterization of the emetic principle present in barley infected with Gibberella saubinetii. Phytopath. 31: 871-885. 1941.
33. Hughes, H. D. Improved method of fighting smut in oats. Iowa Agr. Expt. Sta. Circ. 45. 1918.
34. Kendrick, J. B. Holcus bacterial spot of Zea mays and Holcus species. Iowa Agr. Expt. Sta. Res. Bul. 100. 1924.
35. Kent, G. C. Some physical, chemical and biological properties of a specific bacteriophage of Pseudomonas tumefaciens. Phytopath 27: 871-902. 1937.
36. Kingsolver, C. H. Pathogenicity on Avena and growth response of Pseudomonas coronafaciens (Elliott) Stapp. Ph.D. Thesis, Iowa State Col. Library. 1943.
37. Knaphus, George. Vertical distribution of Pythium graminicola in soil. M.S. Thesis, Iowa State Col. Library. 1951.
38. Litzenberger, S. C. Inheritance of resistance to specific races of crown and stem rust; to Helminthosporium blight, and of certain agronomic characters of oats. Iowa Agr. Expt. Sta. Res. Bul. 370. 1949.
39. McLaughlin, J. H. The effect of soil moisture and temperature upon the prevalence and activity of Pythium in soil. Ph.D. Thesis. Iowa State Col. Library. 1946.
40. McNew, G. L. Crown infection of corn by Diplodia zeae. Iowa Agr. Expt. Sta. Res. Bul. 216. 1937.
41. _____, and D. E. Bliss. Control of yellow-leaf on nursery stock. Iowa Agr. Expt. Sta. Bul. 332. 1935.
42. Meehan, Frances, and H. C. Murphy. A new Helminthosporium disease of oats. Phytopath. 36: 406. (Abst.) 1946.
43. Melhus, I. E. Crown gall of apple nursery stock. Jour. Econ. Ent. 19: 356-366. 1926.
44. _____. A summary of results of cooperative investigations of crown gall in relation to apple nursery stock. Crop Prot. Digest No. 11. 1926.
45. _____. New developments concerning virus diseases of stone fruits. Trans. Iowa Hort. Soc. 76: 133-137. 1941.
46. _____. The late blight outbreak of 1942 and a proposed forecasting service. Trans. Iowa Hort. Soc. 77: 287-293. 1942.
47. _____. Late blight forecasting service. Phytopath. 35: 463-479. 1945.
48. _____., et al. Plant research in the tropics. Iowa Agr. Expt. Sta. Res. Bul. 371. 1949.
49. _____, S. M. Dietz and Florence Willey. Alternate hosts and biologic specialization of crown rust in America. Iowa Agr. Expt. Sta. Res. Bul. 72. 1922.
50. _____, and L. W. Durrell. Studies on the crown rust of oats. Iowa Agr. Expt. Sta. Res. Bul. 49: 1919.
51. _____, J. C. Gilman and J. B. Kendrick. Fungicidal action of formaldehyde. Iowa Agr. Expt. Sta. Res. Bul. 59. 1920.

52. Melhus, I. E., and T. J. Maney. A study of the control of crowngall on apple grafts in the nursery. Iowa Agr. Expt. Sta. Res. Bul. 69. 1921.
53. _____, J. H. Muncie and V. C. Fisk. Grafting as a further means of preventing knots on apple. Phytopath. 18: 127-128. (Abst.) 1928.
54. _____, C. S. Reddy, W. P. Raleigh and L. C. Burnett. Seed treatment for corn diseases. Iowa Agr. Expt. Sta. Circ. 108. 1928.
55. _____, and F. H. Van Haltern. Sclerospora on corn in America. Phytopath. 15: 720-721. 1925.
56. _____, _____, and D. E. Bliss. A study of Sclerospora graminicola (Sacc.) Schroet. on Setaria viridis (L.) Beauv. and Zea mays L. Iowa Agr. Expt. Sta. Res. Bul. 111. 1928.
57. Muncie, J. H. A study of crowngall caused by Pseudomonas tumefaciens on rosaceous hosts. Iowa State Col. Jour. Sci. 1: 67-117. 1926.
58. _____, and M. K. Patel. Studies upon a bacteriophage specific for Pseudomonas tumefaciens. Phytopath. 20: 289-305. 1930.
59. _____, and R. F. Suit. Studies of crowngall, overgrowth and hairy-root on apple nursery stock. Crop Prot. Digest. No. 29. 1930.
60. Murphy, H. C. Physiologic specialization in Puccinia coronata avenae. U. S. Department of Agriculture Tech. Bul. 433. 1935.
61. _____, and L. C. Burnett. Clinton oats arrive. Farm Science Reptr. Vol. 6, No. 4: 3-7. 1945.
62. _____, and Frances Meehan. Reaction of oat varieties to a new species of Helminthosporium. Phytopath. 36: 407. (Abst.) 1946.
63. _____, and T. R. Stanton. Oat varieties highly resistant to crown rust. Jour. Amer. Soc. Agron. 22: 573-574. 1930.
64. _____, _____, and F. A. Coffman. Hybrid selections of oats resistant to smuts and rusts. Jour. Amer. Soc. Agron. 28: 370-373. 1936.
65. Nagel, C. M. Epiphytology and control of sugar beet leaf spot caused by Cercospora beticola Sacc. Iowa Agr. Expt. Sta. Res. Bul. 338. 1945.
66. _____, and G. Semeniuk. Some mold-induced changes in shelled corn. Plant Physiol. 22: 20-33. 1947.
67. Pammel, L. H. Bacteriosis of rutabaga (Bacillus campestris n. sp.) Iowa Agr. Expt. Sta. Res. Bul. 27: 130-135. 1895.
68. _____, C. M. King, and A. L. Bakke. Two barley blights with comparison of species of Helminthosporium on cereals. Iowa Agr. Expt. Sta. Bul. 116. 1910.
69. _____, _____, and J. L. Seal. Corn stalk and corn root diseases in Iowa. Iowa Agr. Expt. Sta. Circ. 21. 1915.
70. _____, _____, and _____. Studies on a Fusarium disease of corn and sorghum. Iowa Agr. Expt. Sta. Res. Bul. 33. 1916.
71. Patel, M. K. An improved method of isolating Pseudomonas tumefaciens Sm. and Town. Phytopath. 16: 577. 1926.
72. _____. Biological studies of Pseudomonas tumefaciens Sm. and Town. and 15 related non-pathogenic organisms. Iowa State Col. Jour. Sci. 3: 271-298. 1929.
73. Platz, G. A. Some factors influencing the pathogenicity of Ustilago zeae (Beckm.) Unger. Iowa State Col. Jour. Sci. 3: 117-214. 1929.
74. _____, L. W. Durrell and Mary F. Howe. Effect of CO_2 upon the germination of chlamydospores of Ustilago zeae. Jour. Agr. Res. 34: 137-147. 1927.
75. Porter, D. R., and I. E. Melhus. The pathogenicity of Fusarium niveum E. F. S. and the development of wilt resistant strains of Citrullus vulgaris Schrad. Iowa Agr. Expt. Sta. Res. Bul. 149. 1932.
76. Porter, R. H. Dry rot of corn and its control. Iowa Agr. Ext. Serv. Bul. 114. 1923.
77. _____. Detection and classification of seed-borne organisms, their effect on germination and their control by seed disinfection in the laboratory and field. Assoc. Off. Seed Anal. of N. Amer. Proc.: 195-213. 1938.
78. _____, and D. V. Layton. Small grain diseases in Iowa and their control. Iowa Agr. Ext. Serv. Circ. 226. 1936.
79. Raleigh, W. P. Infection studies of Diplodia zeae (Schw.) Lev. and control of seedling blight of corn. Iowa Agr. Expt. Sta. Res. Bul. 124. 1930.
80. Reddy, C. S. Resistance of dent corn to Basisporium gallarum Moll. Iowa Agr. Expt. Sta. Res. Bul. 167. 1933.
81. _____. Relation of rate of planting to the effect of corn seed treatment. Iowa State Col. Jour. Sci. 9: 313-324. 1935.

82. Reddy, C. S. Effects of seed treatment on disease-free and diseased seed corn. Phytopath. 26: 105-106. (Abst.) 1936.
83. _____. Flax seed treatment. Phytopath. 26: 106. (Abst.) 1936.
84. _____. Results of corn, oats and flax seed treatments. Annual reports: Northern Iowa Experimental Association, 1934-1950; Southeastern Iowa Experimental Association, 1934-1950.
85. _____, and W. F. Buchholtz. Seed treatment of sugar beets. Iowa Agr. Ext. Serv. Circ. 240. 1937.
86. _____, and W. N. Rice. Seed treatment of good seed corn. Phytopath. 32: 23. (Abst.) 1942.
87. Semeniuk, G. Seedling infection of maize by Diplodia zeae in steamed and non-steamed soil. Phytopath. 30: 22. (Abst.) 1940.
88. _____. Comparative reactions of single crosses of dent maize to Diplodia zeae. Phytopath. 32: 16. (Abst.) 1942.
89. _____. Placement of Diplodia zeae inoculum in the soil in relation to infection of maize seedlings in greenhouse pot experiments. Phytopath. 32: 24. (Abst.) 1942.
90. _____. Seedling infection of dent maize by Sclerotium bataticola Taub. Phytopath. 34: 838-843. 1944.
91. _____, and J. C. Gilman. Relation of molds to the deterioration of corn in storage. A review. Iowa Acad. Sci. 51: 265-280. 1944.
92. _____, C. M. Nagel, and J. C. Gilman. Observations on mold development and on deterioration in stored yellow dent shelled corn. Iowa Agr. Expt. Sta. Res. Bul. 349. 1947.
93. _____, J. R. Wallin, and I. E. Melhus. Root necrosis resistance in maize. Phytopath. 37: 20. (Abst.) 1947.
94. Standen, J. H. Chemical and physical characteristics of maize cobs in relation to the growth of Nigrospora oryzae. Phytopath. 34: 315-323. 1944.
95. _____. Nigrospora oryzae (B. and Br.) Petch on maize. Phytopath. 35: 552-564. 1945.
96. Stanton, T. R., and H. C. Murphy. Oat varieties highly resistant to crown rust and their probable agronomic value. Jour. Amer. Soc. Agron. 25: 674-683. 1933.
97. _____, _____, F. A. Coffman and H. B. Humphrey. Development of oats resistant to smuts and rusts. Phytopath. 24: 165-167. 1934.
98. Stevens, Katherine, I. E. Melhus, G. Semeniuk and Lois Tiffany. A new method of inoculating some Maydeae with Ustilago zeae (Beckm.) Unger, Phytopath. 36: 220-221. (Abst.) 1946.
99. Suit, R. F. Pseudomonas rhizogenes R.B.W.K. and S., its host relations and characteristics. Iowa State Col. Jour. Sci. 8: 131-173. 1933.
100. Summers, T. E. Time and frequency of occurrence of Pythium debaryanum and P. graminicolum on roots of barley. M.S. Thesis, Iowa State Col. Library. 1947.
101. Taylor, G. S., and G. Semeniuk. Guatemalan corn as a source of resistance to Helminthosporium turcicum in maize. Phytopath. 41: 34-35. (Abst.) 1951.
102. Tiffany, Lois H. Delayed sporulation of Colletotrichum on soybeans. Phytopath. 41: 35. (Abst.) 1951.
103. Vestal, E. F., and F. G. Bell. A preliminary study of some environmental factors on the spread of Cercospora leaf spot and yield in checked and drilled sugar beets. Amer. Jour. Bot. 18: 705-716. 1931.
104. Vlitos, A. J., and W. J. Hooker. The influence of sulfur on populations of Streptomyces scabies and other streptomycetes in peat soil. Amer. Jour. Bot. (in press) 1950.
105. Waggoner, P. E. Temperature lapse rates over bare ground and over potato plots on peat and mineral soils. Bul. Amer. Meteor. Soc. 31: 326-329. 1950.
106. _____. Gradients of potato late blight observed around artifically inoculated plants. Phytopath. 41: 36. (Abst.) 1951.
107. _____, and R. H. Shaw. Plant part temperatures influencing the epiphytology of potato and tomato late blight. Phytopath. 41: 36. (Abst.) 1951.
108. Waldee, E. L. Comparative studies of some peritrichous phytopathogenic bacteria. Iowa State Col. Jour. Sci. 19: 435-484. 1945.
109. Wallin, J. R. Parasitism of Xanthomonas translucens (J. J. and R.) Dowson on grasses and cereals. Iowa State Col. Jour. Sci. 20: 171-193. 1946.

110. Wallin, J. R. Seed and seedling infection of barley, bromegrass and wheat by Xanthomonas translucens var. cerealis. Phytopath. 36: 446-457. 1946.
111. _____. A new method of inoculating the Maydeae with smut fungi. Science 109: 312-313. 1949.
112. _____. Forecasting tomato and potato late blight in the north-central region. Phytopath. 41: 37. (Abst.) 1951.
113. _____. Response of the tomato and potato in field plots inoculated with Phytophthora infestans. Phytopath. 41: 37. (Abst.) 1951.
114. _____, and C. S. Reddy. A bacterial streak disease of Phleum pratense L. Phytopath 35: 937-939. 1945.
115. _____, and P. E. Waggoner. The influence of weekly cumulative rainfall and temperature on potato late blight epiphytotics in Iowa. Plant Dis. Reptr. 33: 210-218. 1949.
116. _____, and _____. The influence of climate on the development and spread of Phytophthora infestans in artificially inoculated potato plots. Plant Dis. Reptr. Suppl. 190: 19-33. 1950.
117. Welch, Aaron. Pythium root necrosis of oats. Iowa State Col. Jour. Sci. 19: 361-399. 1945.
118. Welch, A. W., and J. C. Gilman. Hetero- and homo-thallic types of Diaporthe on soybeans. Phytopath. 38: 628-637. 1948.
119. Wilkinson, R. E., and G. C. Kent. Some factors determining the infection of corn by Ustilago zeae (Beckm.) Unger. Iowa State Col. Jour. Sci. 19: 401-413. 1945.
120. Wilson, J. J. The pathological relationship between the host and parasite in varieties and strains of watermelons resistant to Fusarium niveum E. F. S. Iowa Agr. Expt. Sta. Res. Bul. 195. 1936.
121. Yu, T. F. Pathological and physiological effects of Bacillus tracheiphilus E. F. Smith on species of Cucurbitaecae. Nanking Jour. 3: 47-128. 1933.
122. Younkin, S. G. Pythium irregulare and damping off of watermelons. Phytopath. 28: 596. 1938.

IOWA STATE COLLEGE, AMES

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
AGRICULTURAL RESEARCH ADMINISTRATION
UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 204

BIBLIOGRAPHY OF SOYBEAN DISEASES

Supplement 204 June 15, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

BIBLIOGRAPHY OF SOYBEAN DISEASES

Lee Ling

Plant Disease Reporter
Supplement 204 June 15, 1951

INTRODUCTION

This bibliography was initiated while the writer was engaged in studies of soybean diseases in Western China during the Sino-Japanese war. Although all the experimental records were lost, this manuscript survived accidentally. In the writer's sojourn at Washington, D. C. during the past three years, access to the excellent facilities of the Library of the U. S. Department of Agriculture has enabled him to expand the manuscript and to bring it up-to-date. It has also given him the opportunity of checking over many original publications which appeared in various languages. In its present form, this bibliography covers approximately 500 titles published from 1882 to 1950, including a number on soybean diseases in the Orient, which are not easily accessible to western readers. It is recognized, however, that a publication of this nature can never be complete and that a number of papers related to the subject, especially shorter ones, may have been overlooked. The writer would appreciate it if such omissions were brought to his notice.

Following each title in this bibliography, a brief annotation is included to indicate its nature and contents in a general way, but such annotation is not intended to serve as an abstract or a review of the publication concerned. For the convenience of those who may make use of this bibliography, an appendix is also provided, in which the causal agents of all the recorded diseases of soybean are arranged alphabetically according to their nature, with references to all literature citations. Common names and brief descriptions of symptoms are given for the common diseases. For the organisms which were described originally as parasites of soybean, brief diagnoses of their taxonomic characters, taken chiefly from their original descriptions, are also included. Synonyms of the organisms are indicated only in cases where they have appeared in literature concerned with soybean diseases.

The writer wishes to acknowledge his indebtedness to Dr. T. Matsumoto for checking the Japanese literature; to Mr. S. T. Liu for his help in the initial stages of preparation of the manuscript; and to Dr. P. R. Miller for his interest and a review of the manuscript.

ANNOTATED BIBLIOGRAPHY

1. Abramov, I. N. 1931. (Fungal diseases of soybeans in the Far East.) In (Diseases and pests of soybean in the Far East.) pp. 3-84. Far Eastern Sta. Plant Prot., Vladivostock. (In Russian)

An account, chiefly on symptoms and the morphology of causal organisms, of 11 diseases hitherto recorded in Russian Far East. To downy mildew (Peronospora manshurica), varieties with yellow or green beans are susceptible, while those with black and brown beans are practically immune. Sanitation and rotation are recommended for the control of Sclerotinia libertiana, Mycosphaerella phaseolicola may be the perithecial stage of Cercospora daizu. Other diseases include: seedling blight caused by Fusarium sp.; Fusarium wilt; Ascochyta spots caused by A. sojaecola n. sp.; stem break caused by an unidentified fungus; pink efflorescence of pod caused by Fusarium sp.; Septoria leaf spot; leaf spot caused by Isariopsis griseola; and grey mold caused by Hypochnus centrifugus.

2. Adam, D. B., and A. T. Pugsley. 1935. A yellow bacterium associated with 'halo' blight of beans. Australian Jour. Exp. Biol. & Med. Sci. 13: 157-164.

Phytomonas medicaginis phaseolicola occurred occasionally on soybean in Victoria.

3. Adams, J. F. 1923. Plant diseases and their prevalence for 1922 in Delaware. Delaware Sch. Agr. Stencil Circ. 1.

Records the occurrence of a leaf spot caused probably by Bacillus lathyri and of a non-parasitic chlorosis or yellow leaf which was associated with food requirements, especially deficiency of potash.

4. _____. 1925. Department of Plant Pathology. Annual Report Delaware Agricultural Experiment Station for the fiscal year ending June 30, 1924.

Records the occurrence of Bacterium phaseoli sojense.

5. _____. 1926. Department of Plant Pathology. Annual Report Delaware Agricultural Experiment Station for the fiscal year ending June 30, 1926. Delaware Agricultural Experiment Station Bull. 147: 29-35.

Septoria glycines caused a severe defoliation of soybean.

6. _____. 1933. Report of the plant pathologist ofr 1932. Delaware State Board Agr. Quart. Bull. 23: 3-16.

Records the occurrence of Cercospora daizu.

7. Aitken, Y., and B. J. Grieve. 1943. A mosaic virus of subterranean clover. Jour. Australian Inst. Agr. Sci. 9: 81-82.

The mosaic virus of Trifolium subterraneum can infect soybean by artificial inoculation.

8. Albrecht, W. A., and H. Jenny. 1931. Available soil calcium in relation to 'damping off' of soybean seedlings. Bot. Gaz. 92: 263-278.

Damping off is decreased as the availability of calcium increases, while hydrogen-ion concentration is of minor importance in relation to it. Calcium ion is superior to other mono- or divalent ions at equal concentrations. Free, diffusible calcium ion is more effective than absorbed, exchangeable ones.

9. Allescher, A. 1901. Fungi imperfecti. In G. L. Rabenhorst, Kryptogamen-Flora von Deutschland, Oesterreich und der Schweiz, Aufl. 2, Bd. 1, Abt. 6, 1066 pp.

Includes a description of Septoria sojina.

10. Allington, W. B. 1944. Soybean disease investigations at the U. S. Regional Soybean Laboratory. Soybean Dig. 4 (11): 60, 65.

As a result of nursery inspection in 24 States, the following diseases were considered to be of importance: Bud blight, bacterial pustule, bacterial

blight, sclerotial blight, pod and stem blight, downy mildew, and wildfire.

11. Allington, W. B. 1945. Wildfire disease of soybean. Phytopath. 35: 857-869.
Tobacco wildfire was prevalent on soybean. The morphological, physiological, serological, and pathological characters of the isolates of the organism from soybean and tobacco are identical. Water soaking of soybean tissue, especially by beating rain, greatly facilitates its penetration and its spread within the tissue.

12. _____. 1946. Soybean diseases in the corn belt in 1945. Soybean Dig. 6 (11): 48.
An account of the incidence of brown stem rot, bacterial blight, bacterial pustule, and bud blight in the cornbelt.

13. _____. 1946. Bud blight of soybean caused by the tobacco ring-spot virus. Phytopath. 36: 319-322.
Symptoms produced by tobacco ring-spot virus on soybean are described. The identity of the virus was verified by thermal inactivation tests, immunity tests and symptoms.

14. _____. 1946. Brown stem rot of soybean caused by an unidentified fungus. Phytopath. 36: 394.
The symptoms are briefly described. The causal fungus was not fruiting and appeared to be soil-borne.

15. _____, and D. W. Chamberlain. 1948. Brown stem rot of soybean. Phytopath. 38: 793-802.
The causal fungus is named Cephalosporium gregatum n. sp. and its morphology and cultural characteristics are described. The best medium for sporulation was soybean stem agar, on which conidia appeared after 5 days at 20° C. The minimum, optimum, and maximum temperatures for mycelial growth were below 8°, 22-24°, and above 30°; and for germination 15°, 21-25°, and 30° respectively. The optimum and maximum for sporulation were 15-20° and 28°. Air temperatures below 21° were essential for the rapid development of the disease.

16. _____, and _____. 1949. Trends in the population of pathogenic bacteria within leaf tissues of susceptible and immune plant species. Phytopath. 39: 656-660.
The multiplication of Xanthomonas phaseoli and Pseudomonas glycinea within leaves of bean and soybean was initiated about equally in both immune and susceptible hosts. After a time the inhibitory effect of the noncongenial host became apparent and the bacterial population increased less rapidly or decreased markedly. The population within the congenial host continued to increase until destruction of the tissues occurred.

17. _____, and C. V. Feaster. 1946. The relation of stomatal behavior at the time of inoculation to the severity of infection of soybeans by Xanthomonas phaseoli var. sojense (Hedges) Starr and Burk. Phytopath. 36: 385-386.
Experimental results indicate that the bacteria enter the plant when the stomata are open.

18. _____, G. C. Kent, I. W. Tervet, and B. Koehler. 1945. Results of the uniform soybean seed treatment tests in 1944. Plant Dis. Reptr., Suppl. 159: 220-224.
Results of tests with Semesan Jr., New Improved Ceresan, Arasan, and Spergon at 18 locations in U. S. indicate that significant increase in stand were obtained only occasionally and yield was generally unaffected.

19. Allison, J. L. 1947. Present status of soybean diseases. Soybean Dig. 7 (11): 49.
Outlines the organization and scope of the project for soybean disease research started in 1945 by the U. S. Bureau of Plant Industry.

20. Anonymous. 1928. Reports received from Experimental Stations 1926-1927. 251 pp. Empire Cotton Growing Corporation, London.
In Natal, South Africa, soybeans succumbed completely to a Fusarium wilt, and were affected by a bacterial disease, probably caused by Bacterium glycineum.

21. _____. 1930. Research in Botany. North Carolina Agr. Expt. Sta. Ann. Rept. 52: 78-91.
None of the fungicides used to control Cercospora daizu was effective. The fungus overwinters on diseased plant refuse and may survive in the seeds for 3 years.

22. _____. 1932. Virus diseases of tobacco in Nyasaland. Nyasaland Dept. Agr. Bull. 2.
Soybean was infected by a disease similar to leaf curl of tobacco and supported its insect vector, white flies.

23. _____. 1933. Progress made in study of bean diseases. Wisconsin Agr. Exp. Sta. Bull. 425: 101-102.
The robust or yellow strain of bean mosaic can infect soybean.

24. _____. 1934. Plant diseases in Denmark in 1933. Survey of data collected by the State Phytopathological Experiment Station. Tidsskr. Planteavl. 40: 258-300. (In Danish).
Records the occurrence of Bacterium sojae on soybean.

25. _____. 1938. (Summary of the scientific research work of the Institute of Plant Protection for the year 1936. Part III. Virus and bacterial diseases of plants, the biological, the chemical, and the mechanical methods of plant protection). 111 pp. Leningrad. (In Russian)
Bacterium phaseoli var. sojae is included in a list of bacterial pathogens found in USSR.

26. _____. 1943. (Plant diseases in Denmark in 1942. Survey of data collected by the State Phytopathological Experiment Station.) Tidsskr. Planteavl. 48: 1-90. (In Danish).
Records the occurrence of Peronospora manshurica on soybeans.

27. _____. 1943. Soya beans in South Africa. Dept. Agr. South Africa Bull. 240.
Includes a brief account on the following diseases: bacterial blight, Erysiphe polygoni, Peronospora trifoliorum, anthracnose, and Sclerotium rolfsii.

28. _____. 1943. Bacterial diseases increasing on soybean. North Carolina Agr. Exp. Sta. Ann. Rept. 66: 52.
Bacterial pustule disease survives the winter in North Carolina in dead leaves and seeds from diseased plants. The use of resistant varieties and seeds from fields free from the disease are recommended. A second but unidentified bacterial leaf spot was found under some conditions even more destructive than bacterial pustule.

29. _____. 1944. Verslag over de werkzaamheden van den Plantenziekten-kundigen Dienst in het jaar 1942. Versl. Meded. Plantenziektenk. Dienst 103.
Soybean was attacked by Ascochyta sp.

30. _____. 1944. Root rot of snap beans. Georgia Agr. Exp. Sta. Ann. Rept. 56: 57-58.
Diaporthe sojae was found on snap bean, soybean, cowpea and lima bean. Peritheca developed on overwintered stems of all four hosts.

31. _____. 1945. Root rot of snap beans. Georgia Agr. Exp. Sta. Ann. Rept. 57: 49-50.
Inoculation tests indicate that Diaporthe sojae is merely a saprophyte or possibly a weak parasite.

32. Anonymous. 1945. Diseases of field peas, vetches, soybeans, and other forage legumes. Georgia Agr. Expt. Sta. Ann. Rept. 57: 58.
Attempt was made to find soybean varieties resistant to Sclerotium rolfsii by heavy inoculation in the field.

33. _____. 1945. Report of the Federal Experiment Station in Puerto Rico, 1944. 44 pp.
The most important diseases of soybean in Puerto Rico are seed rots and pre-emergence damping-off, which could be effectively prevented by seed treatment with Arasan, Semesan, and Spergon.

34. _____. 1946. Root rot of snap beans. Georgia Agr. Exp. Sta. Ann. Rept. 58: 68-69.
Diaporthe sojae has been found on 12 cultivated plants, including soybean and snap beans.

35. _____. 1946. Diseases of field peas, vetches, soybeans and other forage legumes. Georgia Agr. Exp. Sta. Ann. Rept. 58: 74-77.
Seed treatment failed to increase soybean yield. A mild infection of charcoal rot was found on roots and stem bases of mature soybean plants. The plants are probably susceptible to this disease in the pre-emergence stage. Field observations indicate that Sclerotium rolfsii can travel over or near the surface of soil and attack nearby plants only when conditions, such as continuously high soil moisture content and the presence of abundant organic matter, are extremely favorable for its growth.

36. _____. 1946. Hail damage to soybeans. Soybean Dig. 6 (8): 10-11, 13,
. A progress report on results obtained in 1945. See entry 225.

37. _____. 1947. Diseases of soybeans, Austrian winter peas, vetches and other forage legumes. Georgia Agr. Exp. Sta. Ann. Rept. 59: 83-85.
Field tests failed to reveal any variety of soybean highly resistant to southern blight.

38. _____. 1948. Seed treatment increases yield as well as stand, in 70th Annual Report North Carolina Agricultural Experiment Station. Research and Farming 6 (3): 32-33.
Treatment of soybean seed with Arasan increased the emergence significantly in both greenhouse and field tests. In the field, the increased stands were followed by increases of yields of 3.1, 3.8, and 1.7 bushels per acre respectively for seeding rates of 4, 8, and 12 seeds per foot.

39. _____. 1948. Soybeans respond to dusting for disease control, in 70th Annual Report North Carolina Agricultural Experiment Station. Research and Farming 6 (3): 33.
Six applications of a dust consisting of 7% copper, 3% DDT, 10% wheat flour and 80% Cherokee clay at rates of 30, 60, and 90 pounds per acre reduced bacterial leaf damage about 50 percent and increased yield 5.5 bushels per acre.

40. _____. 1948. Plant Diseases. Notes contributed by the Biological Branch. Agr. Gaz. New South Wales 59: 527-530.
Records mosaic virus and Cercospora daizu on soybean.

41. _____. 1948. Rotation only control for stem rot. Soybean Dig. 8 (10): 18.
Brown stem rot has become increasingly important in the Middle West of the United States. It can be combated by a quadrennial system of crop rotation, the fungus dying out in 3 years in the absence of the host. It is suppressed by temperatures above 80° F, but causes heavy damage between 60° and 70° F. in the late summer and early autumn.

42. Anonymous. 1949. Diseases of field peas, lupines, and soybeans, and lupine seed storage problems. Georgia Agr. Exp. Sta. Ann. Rept. 61: 39-40.
A number of varieties are listed as showing greatest freedom in the field from bacterial pustule, bacterial blight, frog-eye, wildfire, downy mildew, and Septoria leaf spot.

43. Aomori Agr. Exp. Sta. 1937. (Experiments on the prevention of soybean chlorosis.) Jour. Pl. Prot. (Tokyo). 24: 624-625. (In Japanese).
Treating soil with formalin and chloropicrin reduced the disease, but also decreased the yield of soybeans.

44. Appleman, M. D. 1942. Effect of seed treatment on nodulation of soybeans and peas. Proc. Soil Sci. Soc. Amer. 6: 200-203.
Semesan and Ceresan did not decrease nodulation on soybean but Cuprocide prevented it. When seed disinfectants were used, all nodulation was of lateral root type.

45. Armstrong, G. M., and Joanne K. Armstrong. 1948. Nonsusceptible hosts as carriers of wilt Fusaria. Phytopath. 38: 808-826.
Sweet-potato-wilt Fusarium and tobacco-wilt Fusaria were recovered from inoculated soybean which exhibited no external symptoms.

46. _____, and _____. 1949. The Fusarium wilt of cowpeas and soybeans. Phytopath. 39:1.
An abstract of the following entry.

47. _____, and _____. 1950. Biological races of the Fusarium causing wilt of cowpeas and soybeans. Phytopath. 40: 181-193.
Two races of Fusarium oxysporum f. tracheiphilum were differentiated by pathogenicity. Race 1 obtained from both soybean and cowpeas caused wilting of some varieties of both hosts; while race 2, obtained only from cowpeas, caused severe wilting only in some varieties of this host. The cowpea varieties, Lady Finger and Sumptuous, served as excellent differentials for these two races. Lady Finger was killed readily by race 1 but showed practically no symptoms with race 2; whereas Sumptuous reacted vice-versa.

48. Atienza, M. 1927. Sclerotium disease of tomato and pepper. Philipp. Agr. 15: 579-588.
Sclerotium rolfsii isolated from tomato and pepper is pathogenic to soybeans.

49. Atkinson, R. E. 1944. Diseases of soybeans and peanuts in the Carolinas in 1943. Plant Dis. Reptr. Suppl. 148: 254-259.
Records the occurrence and prevalence of the following diseases: Bacterial pustule, bacterial blight, mosaic, frog-eye, anthracnose, downy mildew, Sclerotium rolfsii, Sclerotium bataticola, Fusarium sp. causing root rot, root knot, Nematospora sp. causing yeast spot and two unidentified diseases, namely, leaf spot and white stem spot.

50. _____. 1944. Diseases on soybean in North Carolina. Plant Dis. Reptr. 28: 687.
Bacterial blight, frog-eye, downy mildew, mosaic, and a Phyllosticta leaf spot were found.

51. Atkinson, R. E. 1944. Soybean diseases in Virginia and West Virginia. Plant Dis. Reptr. 28: 1008.
Records the prevalence of the following diseases: Leaf spots caused by Cercospora cruenta and C. canescens, bacterial putsule, downy mildew, frog-eye, mosaic, anthracnose, Phyllosticta leaf spot, and wild-fire.

52. Bain, D. C. 1944. Diseases on soybean in Mississippi. Plant Dis. Reptr. 28: 630.
Records the occurrence of charcoal rot and bacterial pustule.

53. Bain, D. C. 1944. Wildfire and other diseases on soybean in Louisiana. Plant Dis. Reptr. 28: 656.
Records the occurrence of wildfire, stem blight and charcoal rot.

54. _____. 1944. Soybean diseases in Mississippi and Louisiana. Plant Dis. Reptr. 28: 834.
Records the prevalence of bacterial pustule, bacterial blight, southern blight, frog-eye spot, a leaf spot associated with Myrothecium roridum, wildfire, downy mildew, anthracnose, and Diaporthe sojae.

55. Baudys, E. 1931. (Phytopathological notes VII.) Ochrana Rostlin, 11: 178-197. (In Czechoslovakian)
Records the occurrence of soybean mosaic and describes its symptoms.

56. Bazan de Segura, C. 1946. Lista de las principales enfermedades de las plantas determinadas en el Peru por el Departamento de Fitopatologia. Est. Exp. Agr. La Molina Divulg. Agr. 3: 1-13.
Lists under soybean, damping-off caused by Rhizoctonia sp. and powdery mildew caused by Erysiphe polygoni and Oidium balsamii.

57. Berkeley, G. H. 1947. Alfalfa mosaic on pepper in Ontario. Phytopath. 37: 3.
An abstract of the following entry.

58. _____. 1947. A strain of the alfalfa mosaic virus on pepper in Ontario. Phytopath. 37: 781-789.
Alfalfa mosaic virus and its pepper strain and potato-calico strain all produced a mild chlorotic mottle on soybean in inoculation tests.

59. Beltyukova, K. and O. P. Lebedeva. 1936. (On the specialization of Phytomonas tobaca Wolf and Foster on certain host plants.) A. I. Mikoyen Pan-Soviet Sci. Res. Inst. Tob. and Indian Tob. Ind., Krasnodar, Publ. 126: 17-34. (In Russian)
By artificial inoculation the organism can infect soybeans.

60. Bisby, G. R. 1924. The Sclerotinia disease of sunflowers and other plants. Sci. Agr. 4: 381-384.
Sclerotinia sclerotiorum attacked soybeans in Manitoba. The morphology of the fungus is described.

61. Blackie, W. J. 1947. Department of Agriculture. Report for the year 1946. Coun. Paper Fiji 19. 18 pp.
Records the occurrence of an unspecified wilt disease on soybean.

62. Boewe, G. H. 1935. Soybean downy mildew in Illinois. Plant Dis. Reptr. 19: 257-258.
Peronospora manshurica destroyed 10 percent of leaf area of soybeans on average and the incidence was probably increased by heavy mid-June rainfall.

63. Böning, K. 1938. Phyllosticta-Fleckenkrankheit der Sojabohne. Prakt. Bl. Pflanzenb. 16: 168-172.
The symptoms and the morphology of Phyllosticta sojaecola are described. The fungus is seed-borne. Seed disinfection together with the destruction of diseased plants and harvest debris are recommended for its control.

64. Boosalis, M. G. 1947. Necrosis of soybean stem and root caused by Rhizoctonia solani. Phytopath. 37: 3.
Necrotic lesions occur on stem and roots of soybean attacked by Rhizoctonia solani. Staling product of the fungus culture alone reduced the germination of soybean seeds, inhibited the development of secondary roots, caused the necrotic lesions, and killed the seedlings.

65. _____. 1950. Studies on the parasitism of Rhizoctonia solani Kuehn on soybeans. Phytopath. 40: 820-831.

Fourteen isolates of the fungus from soybean and other hosts varied in virulence on soybean, but all the five varieties of soybean were equally susceptible. In soil temperature experiments, two isolates caused most damping-off at 25°-29° C. The diluted filtrate of strongly pathogenic isolate reduced seed germination and inhibited root development in soybean.

66. Bouriquet, G. 1946. Les maladies des plantes cultivées à Madagascar. Encycl. Myc. 12: 1-545. Paris.
The causal organism of a disease killing young soybeans is named Coniothyrium sojae n. sp.

67. Bretz, T. W. 1944. Damping-off and bacterial blight of soybeans in east-central Missouri. Plant Dis. Reptr. 28: 657.
Records the occurrence of the two diseases.

68. _____. 1944. Diseases reported on soybeans. Plant Dis. Reptr. 28: 712.
Records the occurrence of Rhizoctonia, bacterial blight, bacterial pustule, downy mildew, and a wilt caused by Pythium, Fusarium and Macrophomina in southern States.

69. _____. 1944. Diseases observed on soybeans in Missouri. Plant Dis. Reptr. 28: 832-834.
Records the occurrence of the following diseases: Frog-eye leaf spot, downy mildew, charcoal rot, bacterial blight, bacterial pustule, stem and pod blight, bud blight, mosaic, and Alternaria leaf spot.

70. _____. 1944. Summary of plant diseases observed in Missouri during 1943. Plant Dis. Reptr. Suppl. 148: 294-302.
Records the occurrence of Diaporthe sojae, Peronospora manshurica, Pseudomonas glycinea, Xanthomonas phaseoli var. sojense, Macrophomina phaseoli (?), and bud blight (virus ?).

71. Briton-Jones, H. R. and R. E. D. Baker. 1934. Notes on some other fungus diseases in Trinidad. Trop Agr. (Trinidad) 11: 67-68.
Soybeans were attacked by Sclerotium rolfsii.

72. Brundza, K. 1937. Report of the Phytopathological Section of the Plant Protection Station in Lithuania for the year 1935. 32 pp. Kaunas.
Pseudomonas phaseoli var. sojense occurred on soybeans.

73. Burgwitz, G. K. 1925. (Bacterial blight and spotting of soybean (Glycine kispida Maxim.) Morbi Plantarum Leningrad, 14: 38-41. (In Russian with German summary)
Soybean seedlings raised at Leningrad, where the plant was never cultivated before, from seeds received from Mongolia developed a leaf spot caused by Bacterium glycineum. It suggests that the disease must be present in Mongolia and was introduced to Russia with imported seeds.

74. Burkholder, W. H. 1930. The bacterial diseases of the bean. A comparative study. Cornell Agr. Exp. Sta. Mem. 127.
Bacterium vignae var. leguminophila and Bact. viridiflava infected the pods of soybean in inoculation experiments.

75. Butler, E. J. 1918. Fungi and disease in plants. 457 pp. Thacker, Spink & Co.
Includes a description of the downy mildew of soybean and its causal fungus which was said to agree with Peronospora trifoliorum.

76. _____, and G. R. Bisby. 1931. The fungi of India. Imp. Counc. Agr. Res. India Sci. Monogr. 1. 44 pp.
Lists Peronospora trifoliorum, Phyllosticta glycines, and Septoria sojae on soybean. Uromyces sojae previously reported on soybean proved to be Uromyces mucunae on Mucuna.

77. Calhoun, S. 1947. Hail damage to soybeans. Soybean Dig. 7 (9): 14-15.
A report of the work by Kalton and others. See entry 226.

78. Castellani, E. 1948. Le virosi della Soia. Reprinted from Olearia 2: 838-844.
Notes, based largely on literature, are given on the symptoms, manner of spread, and control of soybean mosaic, yellow mosaic, and "curvatura apicale" (bud blight).

79. Celino, M. S. 1936. Diseases of cotton in the Philippines: I. Sclerotium stem rot, with notes on other diseases. Philipp. Agr. 25: 302-320.
Soybean strain of Sclerotium rolfsii appeared to be same as cotton strain.

80. Chamberlain, D. W. 1948. Soybean disease investigations in 1947. Soybean Dig. 8 (10): 18.
1947 presented many reversals in relative importance of soybean diseases. In Illinois, brown stem rot, which was present only in the roots and basal stems, and bud blight caused little damage, whereas Septoria glycines became most destructive. Bacterial blight became more important than bacterial pustule. Wildfire was not observed. Rhizoctonia rot and Alternaria leaf spot were unusually widespread. Of 1,100 introductions, three highly resistant to bacterial blight were singled out as promising.

81. _____, and W. B. Allington. 1948. Effect of temperature on brown stem rot of soybeans. Phytopath. 38: 4.
The disease developed at an air temperature of 15° C., but not at 21° and 27°. The optimum temperature for the growth of the fungus was 22°-24°, for germination of conidia 21°-25° and for spore production 15°-20°.

82. Chamberlain, E. E. 1939. Pea-streak (Pisum virus 3). New Zealand Jour. Sci. Tech. 20A: 365-381.
Soybean is susceptible to this virus.

83. Cherewich, W. J. 1941. Rhizoctonia root rot of sweet clover. Phytopath. 31: 673-674.
A species of Rhizoctonia, probably R. solani, from sweet clover is pathogenic to soybean.

84. Chester, K. S. and W. E. Cooper. 1944. A lethal virus of guar (Cyamopsis psoraloides DC.) Phytopath. 34: 998.
The virus can infect soybeans by inoculation.

85. Chiba Agr. Exp. Sta. 1938. (Experiment on the Prevention of purple spot of soybeans.) Jour. Pl. Prot. (Tokyo) 25: 311. (In Japanese)
Seed treatment with mercuric chloride 1:5,000 for 30 minutes greatly increased the percentage of germination.

86. Ciferri, R. 1927. Notae mycologicae et phytopathologicae. Serie II. No. 5. Riv. Patol. Veg. 17: 209-294.
Soybeans were affected by an Ascochyta morphologically identical with A. pisi except that 20 percent of the spores were uniseptate.

87. Clayton, C. C. 1950. Wildfire disease of tobacco and soybeans. Plant Dis. Reptr. 34: 141-142.
Cross inoculations with the wildfire organisms from tobacco and soybean gave negative results. The soybean isolate caused only slight infection on soybean in 2 out of 22 separate tests.

88. Clinton, G. P. 1916. Notes on plant diseases of Connecticut. Connecticut Agr. Expt. Sta. Ann. Rept. 1915, pp. 421-451.
Records on soybeans a bacterial leaf spot caused by Bacillus sp., a chlorosis, and a crinkling disease.

89. Clinton, G. P. 1934. Plant pest handbook for Connecticut. II. Diseases and injuries. Connecticut Agr. Exp. Sta. Bull. 358: 153-329.
Includes an account of 4 soybean diseases. Bacterium glycineum and Bact. phaseoli var. sojense were believed to be both present in bacterial spot. Gray mold caused by Botrytis cinerea was found to kill the leaves partly. The cause of crinkling chlorosis was not determined.

90. Cobb, G. S., G. Steiner, and F. S. Blanton. 1934. Observations on the significance of weeds as carriers of the bulb or stem nematode in Narcissus plantings. Plant Dis. Reptr. 18: 127-129.
Soybean is listed as one of the plants found infected by Anguillulina dipsaci.

91. Coerper, F. M. 1919. Bacterial blight of soybean. Jour. Agr. Res. 18: 179-193.
A full acoount of the appearance of the disease, and the morphological and cultural characters of the causal organism which was named Bacterium glycineum n. sp.

92. Colwell, W. E. 1945. Fertilizing soys in North Carolina. Soybean Dig. 3: 11-12.
Soybeans grown on the dark, highly organic, poorly drained soils of the North Carolina Lower Coastal Plain suffered from potash deficiency. The application of muriate of potash as top dressing is recommended.

93. Conners, I. L. 1936. Fifteenth Annual Report of the Canadian Plant Disease Survey, 1935. 76 pp.
Records the occurrence of Bacterium glycineum, curly top, and Peronospora manshurica.

94. _____, and D. B. O. Savile. 1943. Twenty-Second Annual Report of the Canadian Plant Disease Survey, 1942. 110 pp.
Records the occurrence of pod and stem blight, and anthracnose.

95. _____, and _____. 1944. Twenty-Third Annual Report of the Canadian Plant Disease Survey, 1943. 122 pp.
Records the occurrence and prevalence of the following diseases: Fusarium oxysporum f. tracheiphilum, Diaporthe sojae, Peronospora manshurica, Phyllosticta sojaecola, Cercospora sojina, and Septoria glycines.

96. _____, and _____. 1948. Twenty-Eighth Annual Report of the Canadian Plant Disease Survey, 1947. 118 pp.
Summarizes the observations on the following soybean diseases: downy mildew, brown stem rot, bud blight, bacterial blight, mosaic and sun scald. Other diseases mentioned include: brown spot, Phyllosticta leaf spot, pod and stem blight, and Fusarium blight.

97. Conover, R. A. 1948. Studies of two viruses causing mosaic diseases in soybean. Phytopath. 38: 724-735.
The symptoms of soybean mosaic, caused by Soja virus 1, and of yellow mosaic, caused by a strain of Phaseolus virus 2, are described. Soybean mosaic was severe at 18.5° C. and largely masked at 29.5°. Soja virus 1 produced systemic infection only on soybeans, but was recovered from the symptomless inoculated leaves of certain varieties of garden beans. The virus was transmitted by the pea aphid and the peach aphid, and also through seeds. The thermal inactivation point was 64-66° C.; longevity in vitro 4-5 days. Yellow mosaic was not markedly affected by air temperature. The virus induced mottling on several other legumes. The thermal inactivation point was 54-56° C.; longevity in vitro 3-4 days. It was not seed-transmitted.

98. Cooper, W. E. 1949. Top necrosis, a virus disease of guar. Phytopath. 39: 347-358.
Soybeans were infected by inoculation with this new virus, producing

a systemic stipple necrosis of the young leaves followed by death of the stem tip.

99. Cox, C. E., and W. F. Jeffers. 1946. Root-knot. Univ. Maryland Ext. Serv. Bull. 113.
Soybean is listed as susceptible to Heterodera marioni.

100. Crall, J. M. 1947. Brown stem rot of soybean in Missouri. Plant Dis. Reptr. 31: 14. 1947.
Records the occurrence and symptoms of the disease. Causal organism unidentified.

101. _____. 1948. Defoliation of soybeans in Southeast Missouri caused by Phyllosticta glycineum. Plant Dis. Reptr. 32: 184-186.
The fungus caused leaf spots, and in severe cases, commonly in association with bacterial blight, killed most leaves. Its morphology was described.

102. _____. 1950. Soybean diseases in Iowa in 1949. Plant Dis. Reptr. 34: 96-97.
Records the distribution, seasonal occurrence, and prevalence of bacterial pustule, downy mildew, bud blight, brown spot, root and stem rot, wildfire, charcoal rot, mosaic, brown stem rot, and stem canker caused by Diaporthe phaseolorum var. batatatis. Varietal susceptibility to bacterial pustule and stem canker is noted.

103. _____, J. C. Gilman, and G. L. McNew. 1949. A study of soybean diseases and their control. Iowa Agr. Exp. Sta. Report on Agricultural Research for the year ending 30 June, 1949. pp. 175-176.
Seed treatment with Arasan, Phygon, and Spergon increased the emergence of Earlyana soybeans in 1948. Survey of the soybean-growing areas showed that the most prevalent disease was brown stem rot. Stem canker was found throughout the State. Phyllosticta glycineum caused premature defoliation and may have reduced yields in some fields.

104. Crandall, B. S. and Javier Dieguez C. 1948. A check list of the diseases of economic plants in the Tingo Maria zone of the Peruvian Montana. Plant Dis. Reptr. 32: 20-27.
Includes Sclerotium rolfsii on soybean.

105. Cromwell, R. O. 1917. Fusarium-blight, or wilt disease of soybean. Jour. Agr. Res. 8: 421-440.
A full account is given of Fusarium blight caused by F. tracheiphilum found in North Carolina. By cultural and morphological studies as well as inoculation experiments, the disease was proved to be identical with cowpea wilt. Largest portion of diseased plants occurred on coarse sandy soil.

106. _____. 1919. Fusarium blight of the soybean and the relation of various factors to infection. Nebraska Agr. Exp. Sta. Res. Bull. 14.
The physical structure of soils under natural conditions is not the limiting factor in the infection of the disease, but acidity under certain conditions has some influence.

107. Dale, W. T. 1943. Preliminary studies of the plant viruses of Trinidad. Trop. Agr. (Trinidad) 20: 228-235.
Soybean appeared to be attacked by common cowpea mosaic in field, and when experimentally inoculated with the virus young soybean seedlings were very seriously infected.

108. Dana, B. F. 1940. Occurrence of big bud of tomato in the Pacific North-West. Phytopath. 30: 866-869.

Soybean was affected by phyllody and aggregation of branches, which is possibly connected with big bud of tomato.

109. Dana, B. F. 1941. Morphological and anatomical features of phyllody in varieties of tomatoes and beans. Phytopath. 31: 168-175.
Includes a description of morphological and anatomical modifications of soybeans affected by phyllody.

110. _____. 1947. Phyllody of common beans, a graft-transmissible disease. Phytopath. 37: 360-361.
Phyllody also occurs on soybean and other hosts, probably due to one or more strains of aster-yellows virus.

111. Darpoux, H. 1945. Contribution a l etude des maladies des plantes oleagineuses en France. Ann. Epiphyt., n.s. 40: 71-103.
A species of Ascochyta, near A. pisi, attacked soybeans slightly. Its morphology is briefly described.

112. Davy, R. H. 1942. Further evidence of the fungicidal value of Spergon. Plant Dis. Reptr. 26: 162-163.
Both Spergon and New Improved Ceresan were found to be effective in the prevention of seed rots and pre-emergence damping-off in soybean.

113. Dennis, R. W. G. and D. G. O'Brien. 1937. Boron in agriculture. West Scot. Agr. Coll. Res. Bull. 5.
Includes a description of boron deficiency of soybean.

114. De Guerpel, H. 1942. Les ennemis et les maladies du Soja. Rev. Bot. Appl. & Agr. Trop. 17: 195-201.
Soybeans in France have so far remained free from diseases. Brief notes are given on following diseases reported in Europe and from America: Heterodera radicicola, H. schachtii, Bacterium glycineum, mosiac, Aecicium glycines, Cercospora cruenta, C. kikuchii, Glomerella cingulata, Hypochnus solani, Sclerotium rolfsii, Sclerotinia libertiana, Septoria glycines, Peronospora manshurica, Phyllosticta sojaecola, Uromyces sojae, and Erysiphe communis.

115. De Turk, E. E. 1941. Plant-nutrient deficiency symptoms in legumes. In Hunger signs in crops. 1st Ed. pp. 241-266. Amer. Soc. Agron. & Nat. Fert. Ass'n., Washington.
Includes descriptions with illustrations of nutritional disorders in soybean due to deficiencies of calcium, iron, magnesium, nitrogen, manganese, and potassium, and toxicities of manganese and zinc.

116. Diachun, S. and W. D. Valleau. 1946. Growth and overwintering of Xanthomonas vesicatoria in association with wheat roots. Phytopath. 36: 277-280.
Xanthomonas phaseoli var. sojense, X. vesicatoria, and Pseudomonas medicaginis var. phaseolicola grew, multilpied, and produced colonies on wheat, tomato, bean and soybean roots, the first named host giving most consistent results.

117. Dickson, J. G. 1947. Diseases of field crops. 420 pp. McGraw-Hill, New York.
Includes a chapter on soybean diseases, giving accounts of the following: Non-parasitic leaf spot and leaf discoloration; mosaic, bud blight; bacterial blight, wildfire; bacterial pustule; Pythium root rot; downy mildew, powdery mildew; anthracnose; Fusarium wilt; Cercospora leaf spot; Septoria brown spot; sclerotial root, stem and crown blights, and brown stem rot.

118. Diehl, W. W. 1946. Microascus trigonosporus from soybean. Plant Dis. Reptr. 30: 426.
Records the isolation of this fungus from yellow soybean seed that came from Albama.

119. Dimmock, F. 1936. Seed mottling in soybeans. Sci. Agr. 17: 42-49.
Both environment and heredity have a definite influence on mottling in soybean seed. The selection of strains possessing a high degree of resistance to mottling is suggested as means of reducing this abnormality.

120. Drayton, F. L. 1926. A summary of the prevalence of plant diseases in the Dominion of Canada 1920-1924. Dom. Can. Dept. Agr. Bull. (n.s.) 71.
Records the occurrence of 3 soybean diseases.

121. Drummond-Goncalves, R. 1941. Mildio em sementes de soja. O Biologico (São Paulo) 7: 238.
Records the occurrence of Peronospora manshurica.

122. Eaton, S. 1935. Influence of sulfur deficiency on the metabolism of the soybean. Bot. Gaz. 97: 68-100.
Soybean plants grown in sulfur deficient solutions were affected by yellowing of leaves, smaller leaflets, and thinner and less succulent stems.

123. Earley, E. B. 1943. Minor element studies with soybeans: I. Varietal reaction to concentrations of zinc in excess of the nutritional requirement. Jour. Amer. Soc. Agron. 35: 1012-1023.
Soybean varieties vary considerably in tolerance to high concentrations of zinc in nutrient solutions.

124. Eckstein, O., A. Bruns, and J. W. Turrentine. 1937. Kennzeichen des Kalimangels. Signes de manque de potasses. Potash deficiency symptoms. B. Westermann & Co., New York.
Includes a description of potash deficiency of soybean.

125. Fenne, S. B. 1942. More about soybean diseases from Virginia. Plant Dis. Reptr. 26: 382.
Bacterial leaf and pod spot, Fusarium wilt, stem rot, root knot, mosaic, and a nutritional deficiency were seen every year.

126. _____. 1949. Alfalfa and soybean diseases in Virginia, 1948. Plant Dis. Reptr. 33: 90-91.
Reports pod and stem blight, frog-eye leaf spot, and sclerotial blight as new invaders. Treatment of low-vitality seed increased the stand very significantly, while treatment of seeds of high germination resulted in very little increase.

127. _____, and W. C. White. 1950. Chemical treatment of soybean seed increases germination in laboratory tests. Plant Dis. Reptr. 34: 206-207.
Seed treatment with Arasan increased the germination 5.6 percent over non-treated seed in average.

128. Frank, A. B. 1882. Gallen der Anguillula radicicola Greef an Soja hispida, Medicago sativa, Lactuca sativa and Pirus communis. Verh. Bot. Ver. Brandenburg, pp. 54-55.
A short description of the root gall.

129. Fuelleman, R. F. 1944. Hail damage to soybeans: Report of 1943 results. Trans. Illinois State Acad. Sci. 37: 25-28.
Field tests on artificial hail damage to Richland soybeans indicate that all rates of defoliation reduce yields severely during the period of pod formation, but only heavy damage affects yield during early growth periods previous to blossoming.

130. Fulton, R. W. 1948. Hosts of the tobacco streak virus. Phytopath. 38: 421-428.
Tobacco streak virus can infect soybean by artificial inoculation.

131. Gardner, M. W. 1924. Indiana plant diseases, 1921. Proc. Indiana Acad. Sci. 33: 163-201.
The causal organism of a bacterial leaf spot of soybean was studied in culture and found to be a non-chromogenic strain of Bacterium glycineum.

132. _____. 1927. Indiana plant diseases, 1925. Proc. Indiana Acad. Sci. 36: 231-247.
Purple seed stain of soybean was found to be due to Cercospora sp., probably C. kikuchii.

133. _____. 1929. Indiana plant diseases, 1927. Proc. Indiana Acad. Sci. 38: 143-157.
Records the occurrence of Diaporthe sojae and Cercospora kikuchii on soybean. Seed infection by the latter fungus appears to emanate often from the hilum.

134. _____, and J. B. Kendrick. 1921. Soybean mosaic. Jour. Agr. Res. 22: 111-114.
A detailed account, chiefly on the symptoms, of the disease.

135. _____, and E. B. Mains. 1930. Indiana plant diseases, 1928. Proc. Indiana Acad. Sci. 39: 85-99.
Records the occurrence of downy mildew on soybean.

136. Gaumann, E. 1923. Beitrage zu einer Monographie der Gattung Peronospora Corda. Beitr. Kryptogamenflora Schweiz. 5: 1-360.
Contains a technical description of Peronospora manshurica.

137. Geeseman, G. E. 1950. Physiologic races of Peronospora manshurica on soybeans. Agron. Jour. 42: 257-258.
Three races were differentiated on 7 pure line varieties of soybean. Richland was susceptible to races 1 and 2, and when inoculated with race 3, developed resistant pinpoint type of lesions. Chief, Manchu 3, T 117, Mukden and Dunfield were immune to races 1 and 3, and formed small, resistant-type lesions with race 2. Illini was susceptible to all races.

138. Gibbons, F. P., and E. L. Nixon. 1929. The invasion of plant tissue by bacterial parasites. Ann. Rept. Pennsylvania Agr. Exp. Sta. 42: 14-15.
Bacterium leguminosarum enters the cells of soybean through cavities in the walls. It migrates in its early invasion as inter- and intracellular zoogloeae, while in later stages it becomes intra-cellular.

139. Gibson, F. 1922. Sunburn and aphid injury of soybeans and cowpeas. Arizona Agr. Exp. Sta. Tech. Bull. 2.
A weakly parasitic Alternaria infected soybean leaves through aphids and sunburn injuries. The fungus is briefly described and named A. atrans n. sp.

140. Gilman, J. C., and L. H. Tiffany. 1950. Some species of Colletotrichum on leguminous forage crops. Phytopath. 40: 10.
Of the 3 cultures isolated from soybean, one is the conidial stage of Glomerella glycines (not Colletotrichum glycines), one similar to Colletotrichum glycines and C. truncatum, and the third resembles C. pisi.

141. Ginsburg, J. M. 1925. Composition and appearance of soybean plants grown in culture solutions each lacking a different essential element. Soil Sci. 20: 1-13.
Pathological conditions due to the lack of any one element appeared first and most pronounced in plants grown in the calcium-free solution, followed in order in the plants grown in solutions without nitrogen, potassium, magnesium, sulfur, iron, and phosphorus. The nature of the injury sustained and the appearance of the plants are described.

142. Godfrey, G. H. 1929. A destructive root disease of pineapples and other plants due to Tylenchus brachyurus n. sp. Phytopath. 19: 611-629.
Soybean was infected by the nematode in Hawaii.

143. Goodey, T. 1940. The nematode parasites of plants catalogued under their hosts. 80 pp. Imp. Bur. Agr. Parasit.
Lists soybean as the host of Anguillulina dipsaci (leaf gall), A. pratensis, Heterodera schachtii, and H. marioni.

144. Goot, P. van der, and H. R. A. Muller. 1932. Plagen en ziekten der kedelee op Java. Beknopt voorloopig oversicht. (Pests and diseases of the soybean crop in Java. Preliminary report.) Landbouw Tijdschr. Vereen. Landb. Nederl.-Indie. 7: 683-704. (English summary in p. 758-759).
Diseases are of minor importance in Dutch East Indies. Foot rot (Sclerotium rolfsii) may cause loss under wet conditions. Anthracnose under prevailing wet weather may cause leaf drop and seed rot. Seedlings from infected seeds may die. Slime disease caused by Bacterium solanacearum is also reported.

145. Goto, K. 1925. (Miscellaneous notes on the plant diseases found in Morioka. I.) Jour. Pl. Prot. (Tokyo) 12: 677-682. (In Japanese).
Records the occurrence of a Phomopsis on soybean pods. The fungus resembles the imperfect stage of Diaporthe sojae.

146. _____. 1937. (Acceleration of growth in the light case of soybean purple seed. Preliminary report.) Sci. Bull. Alumni Ass'n. Morioka Coll. Agr. & For. 13: 1-14. (In Japanese)
Soybean seeds lightly infected by Cercosporina kikuchii grow faster than the healthy ones.

147. _____, and K. Takahashi. 1926. (Miscellaneous notes on the plant diseases found in Morioka II.) Jour. Pl. Prot. (Tokyo) 13: 154-158. (In Japanese).
Records the occurrence of a new stem blight caused by Peckia sp. The symptoms and the morphology of the fungus are briefly described.

148. Groves, J. W. and A. J. Skolko. 1945. Notes on seed-borne fungi. III. Curvularia. Canad. Jour. Res. Sect. C, Bot. Sci. 23: 94-104. 1945.
Curvularia trifolii was isolated from soybean seed, but appeared to have no pathological significance.

149. Guerpel, H. 1937. Les ennemis et les maladies du soja. Rev. Bot. Appl. 17: 195-201.
A brief review of some insect pests and diseases affecting soybeans.

150. Haenseler, C. M. 1946. Soybean diseases in New Jersey. Plant Dis. Notes, New Jersey Agr. Exp. Sta. 23: 17-20.
Pod and stem blight causes a marked reduction in yield due to premature killing of the plants. Mosaic and downy mildew are of rare occurrence. Purple seed disease was proved to be caused by a fungus.

151. _____. 1947. Pathologist describes four principal diseases of field soybeans in Jersey. New Jersey Agr. 29: (3) 4.
A popular brief account on the effects of mosaic, downy mildew, pod and stem blight, and purple seed disease caused by an unspecified fungus.

152. Hagedorn, D. J. 1950. A cucumber virus strain with a wide leguminous host range. Phytopath. 40: 11.
A new strain of cucumber virus 1 can infect soybean.

153. _____, and J. C. Walker. 1949. Wisconsin pea streak. Phytopath. 39: 837-847.
Soybean was infected by inoculation with the virus, producing very faint general chlorosis.

154. Hagedorn, D. J., and J. C. Walker. 1950. The relation of bean virus 2 to pea mosaic in Wisconsin. Phytopath. 40: 684-698.
Four isolates of bean virus 2 from pea can all infect soybean.

155. Hansford, C. G. 1934. Annual report of Mycologist, 1933. Dept. Agr. Uganda Ann. Rept. 1933, part 2, pp. 48-51.
Soybean showed a mosaic condition unaccompanied, however, by any diminution in yield.

156. Hanson, E. W. 1938. Parasitism and physiologic specialization in Fomes lignosus. Phytopath. 28: 8.
Fomes lignosus is pathogenic to soybeans by artificial inoculation.

157. Hara, K. 1915. (Spot disease of soybean.) Agr. Country 9: 28. (In Japanese).
A brief description of a new leaf spot disease of soybean caused by Cercospora sojina n. sp.

158. _____. 1918. (Diseases of soybean.) Agr. Country 12: 18. (In Japanese).
Describes a pod blight caused by Fusarium roseum ? and a leaf spot caused by Ascochyta sp.

159. _____. 1928. (Notes on fungi of eastern Asia) Bull. Agr. Ass'n. Shizuoka No. 360, appendix. (In Japanese)
Includes a short note on Mycosphaerella sojae found in China.

160. _____. 1930. Pathologia agriculturalis plantarum. 950 pp. Tokyo. (In Japanese)
Includes descriptive accounts of the following soybean diseases: Peronospora manshurica, Cercospora sojina, Colletotrichum glycines, Gloeosporium sp., Septoria glycines, Ascochyta sp. causing leaf spot, Phakopsora pachyrhizi, Fusarium roseum, Bacterium glycines, Bact. sojae var. japonicum, Ascochyta sp. causing pod blight, Hypochnus centrifugus, Cercosporina kikuchii, brown spot of seeds of unknown cause, Macrophoma mame, Mycosphaerella sojae, Phyllosticta sojaecola, Ascochyta sojae, Phomopsis sojae, Peckia sp., Ophionectria sojae, mosaic, Heterodera schachtii, and Cuscuta chinensis.

161. Harris, M. R., and C. W. Ellett. 1945. A Penicillium disease of soybeans. Phytopath. 35: 144-145.
An undetermined species of Penicillium was isolated from two types of lesions on soybeans in Ohio. Inoculation of seeds with the mold, in addition to the production of lesions on hypocotyls and cotyledons, resulted in retarded germination, stunting of the seedlings, distortion and reduction of the size of leaves, and sometimes in the development of lateral buds. Preliminary results of varietal susceptibility trials are given.

162. Haskell, R. J. 1926. Diseases of cereal and forage crops in the United States in 1925. Plant Dis. Reptr., Suppl. 48: 301-381.
Includes records on the prevalence of the following soybean diseases: Bacterial blight, bacterial pustule, Cercospora leaf spot, and Septoria brown spot.

163. _____, and J. I. Wood. 1923. Diseases of cereal and forage crops in the United States in 1922. Plant Dis. Reptr., Suppl. 27: 164-265.
Records the occurrence of Peronospora sp. on soybean.

164. Heald, F. D. 1906. New and little-known plant diseases in Nebraska. Science, n. s. 23: 624.
One of the first reports on bacterial blgiht.

165. _____. 1906. Report on the plant diseases prevalent in Nebraska during the seasor of 1905. Nebraska Agr. Exp. Sta. Ann. Rept. 19: 20-81.

Records the occurrence of bacterial blight caused by Bacillus sp. with recommendations on its control.

166. Hedges, F. 1922. Bacterial pustule of soybean. Science, n. s. 56: 111-112.
A leaf spot of soybean which differs from the bacterial blight in the earlier stage of attack, was found in Washington, D. C. The organism, which is named Bacterium phaseoli var. sojense n. var. without description, resembles Bact. phaseoli but differs slightly in growth characters.

167. _____. 1924. Soybean pustule. Comparative studies with Bacterium phaseoli var. sojense Hedges and Bacterium phaseoli E. F. S. Phytopath. 14: 27-28.
A condensed report of the following entry.

168. _____. 1924. A study of bacterial pustule of soybean, and comparison of Bacterium phaseoli sojense Hedges with Bacterium phaseoli E. F. S. Jour. Agr. Res. 29: 229-251.
A detailed account on the history and symptoms of the bacterial pustule and varietal susceptibility to it. The pustules are caused by both hypertrophy and hyperplasia chiefly of parenchyma. On Phaseolus, Bacterium phaseoli sojense does not form pustules and is less infectious than Bact. phaseoli. To soybeans, Bact. phaseoli is very weakly pathogenic. With the exception of the colonies on beef agar plates, the two organisms behave alike on all the cultural media tested.

169. _____. 1926. Bacterial wilt of beans (Bacterium flaccumfaciens), including comparisons with Bacterium phaseoli. Phytopath. 16: 1-22.
Bacterium flaccumfaciens infected soybeans by artificial inoculation.

170. Heinze, K. 1941. Feldinfektionsversuch mit dem Sojabohnenvirus. Mitt. Biol. Reichsanst. f. Land u. Forstw. 65: 23.
The Giessen and Dieckmann 1940 selections of soybean are resistant to mosaic.

171. _____. 1942. Die Feldbereinigungen bei Sojakulturen als Schutzmassnahme gegen die Ausbreitung des Virösen Sojamosaiks. Vorl. Mitt. Züchter 14: 254-258.
Mosaic, the most destructive disease of soybean in Germany, needs stringent field sanitation to prevent the perpetuation of the virus through seeds. In the experimental fields, a marked difference was found between the plots from which the infected plants had been eliminated and the untreated controls.

172. _____, and E. Kohler. 1940. Die Mosaikkrankheit der Sojabohne und ihre Übertragung durch Insekten. Phytopath. Zeitschr. 13: 207-242.
A mosaic disease of soybean identical or closely related to that described in North America was found in Germany. The virus is transmitted by seeds, by sap, and by the following insects, Doralis frangulae, D. rhamni, D. fabae, Macrosiphum solanifolii, Myzus ornatus, Neomyzus circumflexus, Anlacorthum pseudosolani, and Myzodes persicae. The virus in sap is inactivated at a temperature of 61° C., and remains viable for 3 to 4 days at 21-23° C. The virus is transmissible to beans and vetches. Methods of control are suggested.

173. Hemmi, T., 1915. (On a new brown spot disease of soybean) Bull. Hokkaido Agr. Ass'n. 5 (4): 1-4. (In Japanese)
A condensed report of the following entry.

174. _____. 1915. A new brown-spot disease of the leaf of Glycine hispida Maxim. caused by Septoria glycines sp. n. Trans. Sapporo Nat. Hist. Soc. 6: 12-17.
Describes the symptoms of the disease and the morphology of the causal fungus. The differences between Septoria glycines and S. sojina are tabulated.

175. Hemmi, T. 1920. Beiträge zur Kenntnis der Morphologie und Physiologie der Japanischen Gloeosporien. Jour. Coll. Agr. Hokkaido Imp. Univ. 9: 1-159.
Describes the morphology of Colletotrichum glycines and Gloeosporium sp. on soybean pods.

176. _____. 1921. Nachtrage zur Kenntnis der Gloeosporien. Jour. Coll. Agr. Hokkaido Imp. Univ. 9: 305-346.
Cultural studies on the influence of sulfuric acid, boric acid, sodium hydroxide, and different concentrations of sugar and pepton on the growth of Gloeosporium sp. and Colletotrichum glycines.

177. _____. 1940. Studies on septorioses of plants. VI. Septoria glycines Hemmi causing the brown-spot disease of soybean. Mem. Coll. Agr. Kyoto Imp. Univ. 47: 1-14.
The morphological and physiological characters of Septoria glycines are described. Pycnospores germinated readily within 24 hours between 16° and 36° C., with greatest expansion of germ-tubes at 28° C. Growth took place at temperatures 5°-36° C., with optimum at 24°-28° C. Formation of pycnospores was accelerated by the fluctuation of temperature, but was not affected by light. The fungus was transmitted through infected seeds and contaminated soil.

178. Hendrickx, F. L. 1939. Observations phytopathologiques à la station de Mulungu en 1938. In Rapport annuel pour l'exercice 1938 (2ième partie). Publ. Inst. Nat. Étud. Agron. Congo Belge, pp. 117-128.
Records the occurrence of Ascochyta sojaecola on soybean in Belgian Congo.

179. Heuberger, J. W. 1945. Department of Plant Pathology. In Delaware Agr. Exp. Sta. Rept. 1944-45, pp. 33-39. Delaware Agr. Exp. Sta. Bull. 259.
Dithane applied as a soil disinfectant at 100 lbs. per acre at planting time severely damaged the soybean seeds. Spergon is considered the best for seed treatment of soybean.

180. _____, and T. F. Manns. 1943. Effect of organic and inorganic seed treatments on rate of emergence, stand, and yield of soybeans. Phytopath. 33: 1113.
An abstract of the following entry.

181 _____, and _____. 1944. Effect of organic and inorganic seed treatments on rate of emergence, stand, and yield of soybeans. Delaware Agr. Exp. Sta. Pamp. 11. (Mimeographed.)
Arasan, Spergon, Ceresan, and Dow No. 5 accelerated emergence of seedlings and increased stand, Arasan being the only material which increased the yield significantly.

182. Hildebrand, A. A. 1942. Diseases of soybeans, their control. Canad. Hort. and Home Mag. 69: 129-131, 142.
A popular account on the control of soybean diseases by seed treatment, and cultural practices, and other precautions.

183. _____. 1944. In Symposium of seed-borne diseases. Proc. Canad. Phytopath. Soc. 12: 18-21.
Soybeans are susceptible to at least 32 parasitic diseases, and 11 of a reported total of 13 seed-borne diseases occur in Canada. Mosaic, pod and stem blight, and anthracnose are not amenable to control by surface disinfectants, but downy mildew and brown spot can be effectively controlled by seed treatment. Treatment of mildewed seeds with Spergon and Arasan increased emergence, and reduced post-emergence diseases.

184. _____. 1948. Keeping abreast with soybean diseases in Ontario. Soybean Dig. 8 (10): 16-17.
Since 1942, 14 diseases have been found. Eight or nine of them are seed borne. Mosaic and pod and stem blight were severe in 1942.

Fusarium blight was prominent in 1943 and has been on the increase since. Bud blight and charcoal rot first appeared in 1944. In 1945-46 downy mildew was exceptionally widespread, and infection was largely disseminated by contaminated seeds. Other diseases reported are Sclerotinia stem rot and brown stem rot. Treatment with Spergon of seed encrusted with downy mildew increased the yield in 1947.

185. Hildebrand, A. A. 1948. An occurrence of brown stem rot of soybeans in Ontario. Sci. Agr. 28: 261-263.
The fungus is soil-borne and infection takes place either through the roots or at the base of the stem near ground-level. The incidence of the disease depends upon low air temperature.

186. _____, and L. W. Koch. 1945. Some studies on Macrophomina phaseoli (Maubl.) Ashby in Ontario. Sci. Agr. 25: 690-706.
Ontario isolate of Macrophomina phaseoli from soybean and Texas isolate from cotton may be distinguished by the differences in the size and number of sclerotia produced in culture.

187. _____, and _____. 1946. Seed treatment and other tests with soybeans in Ontario. Phytopath. 36: 401.
An abstract in the following entry.

188. _____, and _____. 1947. Soybean diseases in Ontario and effectiveness of seed treatment. Phytopath. 37: 111-124.
Of the 13 parasitic diseases known to occur in Canada, 8, or possibly 9 are seed-borne. In a 3-year experiment of seed treatment with Spergon, Arasan and Fermate, increase in emergence and yield was obtained only with poor quality, weather-damaged or cracked seeds treated with Spergon. In no other cases were the increases in early stands correlated with significant gains in yield. Varietal differences were noticed in the susceptibility to Septoria glycines.

189. _____, and _____. 1947. Observations on bud blight of soybeans in Ontario. Sci. Agr. 27: 314-321.
The symptoms of the disease are described. Little evidence was obtained on the seed-borne nature of the virus.

190. Hildebrand, A. A., and L. W. Koch. 1950. Observations on six years' seed treatment of soybeans in Ontario. Sci. Agr. 30: 112-118.
From 1943 to 1948, soybean seed of the variety A. K. Harrow was treated with Spergon, Arasan, Fermate, Phygon, Phygon-XL and F-800. Only in the case of seed of extremely poor quality in 1943 treatment with Spergon increased both emergence and yield.

191. Hiratsuka, N. 1932. (Notes on soybean rust) Trans. Biol. Soc. Tottori, 1: 8-11. (In Japanese)
The name Phakopsora pachyrhizi is adopted for the fungus, with P. vignae and P. sojae as its synonyms.

192. _____. 1935. Uredinales collected in Korea I. Bot. Mag. (Tokyo). 49: 145-152.
Includes Phakopsora pachyrhizi on soybean.

193. _____. 1935. Phakopsora of Japan I. Bot. Mag. (Tokyo). 49: 781-788.
Includes Phakopsora pachyrhizi on soybean with a discussion of its synonyms.

194. _____, and Y. Hashioka. 1933. Uredinales in Formosa I. Trans. Tottori Soc. Agr. Sci. 4: 156-165.
Includes Phakopsora pachyrhizi on soybean.

195. Hokkaido Agr. Exp. Sta. 1922. (Investigations on chlorosis of soybeans.) Jour. Pl. Prot. (Tokyo). 9: 101-105. (In Japanese)
The history of the study of the disease was traced back to Frank in 1881. The causal organism was identified as Heterodera schachtii. The susceptibility of Japanese varieties of soybean to the disease were recorded. The disease was prevalent especially under the following conditions: (1) Soybean rotated with other susceptible crops; (2) on soils with inadequate fertilizers; and (3) on dry, sandy soils with poor water preserving capacity.

196. Holbert, J. R., and W. L. Burlison. 1931. Cold injury. Phytopath. 21: 128.
Three varieties of soybeans, Wilson, Virginia, and Illini differed greatly in susceptiblity to cold injury.

197. Holdeman, Q. L. 1950. Some falcate-spored Colletotrichums on legumes. Phytopath. 40: 12-13.
A number of falcate-spored species of Colletotrichum isolated from Lima bean, soybean, red clover, and Lotus spp., while differing in cultural characters and pathogenicity, were considered all as strains of one species, probably C. truncatum.

198. _____. 1950. Pod- and seed-spotting of soybeans caused by Helminthosporium vignicola. Phytopath. 40: 788.
Only the title is given.

199. Honey, E. E. 1944. Brown spot on soybean in Wisconsin. Plant Dis. Reptr. 28: 656.
Records the occurrence of the brown spot disease.

200. _____. 1944. Soybean diseases in Wisconsin. Plant Dis. Reptr. 28: 871-872.
Light infections of bacterial blight, brown spot, mosaic, bud blight, and downy mildew were observed.

201. _____, J. G. Dickson, and F. R. Jones. 1944. Diseases of soybeans in Wisconsin. Plant Dis. Reptr. 28: 988-990.
Records the occurrence of bacterial blight, downy mildew, brown spot, bud blight, mosaic, and a root rot of undetermined cause.

202. Hooker, W. J. 1947. Parasitism of Actinomyces scabies on various plants. Phytopath. 37: 10.
An abstract of the following entry.

203. _____. 1949. Parasitic action of Streptomyces scabies on roots of seedlings. Phytopath. 39: 442-462.
In soil agar, the fungus caused severe necrosis, especially at the root tips, and generally precluded the development of lateral roots in soybean and several other plants tested.

204. Hopkins, E. W. 1933. Leaf-wrinkle, a nutritional disorder of soybean. Pl. Physiol. 8: 333-336.
The disorder seems to arise from a complex relation of K, Ca, Mg, and N. The symptoms are described.

205. Hopkins, J. C. F. 1931. Plant pathology in Southern Rhodesia during the year 1930. Rhodesia Agr. Jour. 28: 384-389.
Records the occurrence of Macrophomina phaseoli and Helicobasidium on soybeans.

206. Hori, S. 1915. (Phytopathological notes V. Sick soil of soybean caused by nematodes.) Jour. Pl. Prot. (Tokyo) 2: 927-932. (In Japanese).
Heterodera sp. was found in soils causing a serious disease of soybean. Soil treatment with calcium cyanamide is recommended.

207. Howard, F. L. 1942. An undetermined, apparently virus, disease of edible soybean in Rhode Island. Plant Dis. Reptr. 26: 381.
An outstanding crinkle or rugose symptom was found on all of 21 varieties grown in the experimental plot.

208. Hsu, J. S. 1944. Variability of monoconidial cultures of Glomerella glycines. Chinese Jour. Exp. Biol. 2: 13-22.
Monoconidial strains of the fungus were found to vary in morphology, growth rate, reaction to temperature and acidity, sugar requirements, and pathogenicity.

209. Ikeno, S. 1933. Studies on Sclerotium disease of the rice plant. VIII. On the relation of temperature and period of continuous wetting to the infection of soybean by the sclerotia of Hypochnus sasakii Shirai and on autolysis of the same fungus. Forsch. Geb. Pflanzenk. (Kyoto) 2: 238-256.
The minimum periods of continuous wetting necessary for the infection of soybean were found to be about 24 hours at 24° and 18 hours at 28° and 32° C. With injured soybean leaves the minimum periods are 24 hours at 20° and 24°, 18 hours at 28°, and 12 hours at 32° C.

210. Ishiyama, T. 1936. New or noteworthy fungi parasites on agricultural plants in southern Saghalien. Trans. Sapporo Nat. Hist. Soc. 14: 297-308.
Includes a description of Ascochyta sojaecola.

211. Ito, S. 1921. (Investigations on soybean chlorosis). Hokkaido Agr. Exp. Sta. Rept. 11: 47-59. (In Japanese)
Similar to entry 195.

212. _____. 1936. Phycomycetes. -- Mycological Flora of Japan. 1: 1-205. (In Japanese).
Includes a description of Peronospora manshurica.

213. _____. 1941. Uredinales -- Mycological Flora of Japan. 2 (2): 1-141. (In Japanese)
Includes a description of Phakopsora pachyrhizi.

214. Iwadare, S. et al. 1943. (A list of the diseases of cultivated plants in Manchuria.) Manchuria Agr. Exp. Sta. Rept. 45: 1-223. (In Japanese)
The following diseases of soybeans are described: Heterodera schachtii, Bacterium glycineum var. japonicum, B. phaseoli var. sojense, Peronospora manshurica, Septoria glycines, Cercospora sojina, Pleosphaerulina sojaecola, Macrophoma mame, Mycosphaerella sojae, Colletotrichum glycines, Uromyces sojae, Phakopsora pachyrhizi, Gibberella sp., Ascochyta sojae, Sclerotinia libertiana, Cercosporina kikuchii, mosaic, Cuscuta chinensis, and brown spot of seed of unknown cause.

215. Johnson, A. G., and F. M. Coerper. 1917. A bacterial blight of soybean. Phytopath. 7: 65.
Reports a bacterial blight of soybean under investigation in Wisconsin.

216. Johnson, F. 1943. Soybean streak in Ohio. Plant Dis. Reptr. 127: 86-87.
The disease is characterized by a bronzing of the foliage and numerous, small, necrotic streaks on growing-tips. Inoculation to cowpea and tobacco indicates that the causal virus belongs to the group of tobacco ring-spot viruses.

217. Johnson, H. W. 1950. Plant disease research on forage crops in the Bureau of Plant Industry, Soils, and Agricultural Engineering. Plant Dis. Reptr. Suppl. 191: 42-59.
Soybean disease research was reviewed, with special reference to studies on brown stem rot, testing resistance of varieties to 3 bacterial foliage diseases, seed treatment, copper dusting, and studies on Diaporthe, Macrophomina phaseoli, downy mildew, and Cercospora kikuchii.

218. Johnson, H. W., and B. Koehler. 1943. Soybean diseases and their control. U. S. Dept. Agr. Farmers' Bull. 1937.
Brief accounts are given of following diseases: bacterial blight, bacterial pustule, pod and stem blight, frog-eye spots, brown spot, anthracnose, downy mildew, Alternaria leaf spot, arsenical injury, mosaic, chlorosis due to deficiencies of potash, iron, and nitrogen, seed discolorations associated with Cercospora and Alternaria, charcoal rot, sclerotial blight, Fusarium blight, root rots, and lightning injury.

219. _____, and C. L. Lefebvre. 1942. Downy mildew on soybean seeds. Plant Dis. Reptr. 26: 49-50.
Soybean seeds infected by downy mildew appeared to be covered with a milky-whitish crust and the coats were crinkled and cracked.

220. Johnson, J. 1916. Host plants of Thielavia basicola. Jour. Agr. Res. 7: 289-300.
Soybean is included as a host.

221. _____. 1922. The relation of air temperature to the mosaic disease of potatoes and other plants. Phytopath. 12: 438-440.
Soybean mosaic is inhibited at temperatures from 26° to 28° C.

222. _____. 1939. Studies on the nature of brown root rot of tobacco and other plants. Jour. Agr. Res. 58: 843-863.
Soybean when grown in soil infested by tobacco brown root rot was susceptible to the disease.

223. _____. 1942. Studies on the viroplasm hypothesis. Jour. Agr. Res. 64: 443-454.
Soybean is susceptible to a new bean yellow necrosis virus by artificial inoculation.

224. Jones, F. R. and J. H. Torrie. 1946. Systemic infection of downy mildew in soybean and alfalfa. Phytopath. 36: 1057-1059.
Planting of soybean seeds encrusted with oospores of Peronospora manshurica gave rise to seedlings with lesions apparently connected by mycelium in a systemic infection.

225. Kalton, R. R., and J. C. Eldredge. 1946. The effect of simulated hail injury. Soybean Dig. 6 (3): 14-15.
Effects of the damage simulating hail injury on soybean were tested by removing artificially leaves and parts of the stems. Reductions in yield varied with the degree of damage and, for any one degree of damage, with the stage of plant development. Time of maturity and plant height were affected at earlier stages of development, while seed quality, oil content and iodine number were most significantly affected when the damage was inflicted during and after pod formation.

226. _____, C. R. Weber, and J. C. Eldredge. 1949. The effect of injury simulating hail damage to soybeans. Iowa Agr. Exp. Sta. Res. Bull. 359: 736-796.
Damages simulating light, medium, and heavy hail injuries were inflicted by clipping and beating on soybeans in different stages of development. Yields were most reduced when the damage was inflicted as seed development began in the lower pods, and were least reduced when the plants were 6 to 12 inches tall with 2 to 5 trifoliate leaves unrolled. Injuries before and during blooming delayed maturity, while injury after the "green bean" stage hastened maturity. Medium and heavy damages during seed development lowered its quality and reduced its size.

227. Kataeva, O. 1932. (Diseases of Soy) in (Summary account of Mountain Zonal Maize-Soy-Potato Experiment Station for 1931. Part III. Department of Phytopathology -- Diseases of maize and soy under conditions of North Caucasus). Nauchn. Trudy Gorskaia

Zonal. Kukur-Soevo-Kartof. Opytn. Stan. Ser. 1: 4: 79-101. (In Russian).
Twenty-two fungi and bacteria were found on soybean. Descriptions of symptoms and causes are given, with illustrations, for the following diseases: seedling blight caused by Fusarium sp.; Fusarium wilt; a stem disease caused by Gibberella sp.; bacterial blight; bacterial pustule; Phyllosticta leaf spot; stem breaking caused by Fusarium sp.; downy mildew; sclerotiniosis; and mosaic. Methods of prevention and control are briefly described.

228. Katsufuji, K. 1919. Yellow dwarf, a new nematode disease of soybean. Ann. Phytopath Soc. Japan. 1: 12-16.
The symptoms of the disease are described and the pathogene is attributed to Heterodera schachtii.

229. Kawatsuka, K. 1920. (Bacterial disease of soybean.) Jour. Pl. Prot. (Tokyo) 7: 220-221. (In Japanese)
A review of entry 91.

230. Kendrick, J. B., and M. W. Gardner. 1921. Seed transmission of soybean bacterial blight. Phytopath. 11: 340-342.
The infection may be carried externally or internally with the seeds which sometimes show lesions themselves.

231. _____, and _____. 1924. Soybean mosaic: seed transmission and effect on yield. Jour. Agr. Res. 27: 91-98.
The disease is transmitted by the seed, usually in rather low percentage varying with the varieties. Selecting seeds from healthy plants as a control measure was proved effective. Mosaic has little influence on the germination power of the seeds but caused a loss of 30 to 75 percent in yield.

232. Kent, G. C. 1942. Soybean diseases in Iowa. Plant Dis. Reptr. 26: 359.
Records the prevalence of root necrosis caused by Pythium spp. and Rhizoctonia, bacterial blight, and mosaic.

233. _____. 1945. A study of soybean diseases and their control. Iowa Agr. Exp. Sta. Report on Agricultural Research for the year ending June 30, 1945. Part I, pp. 221-222.
Increase in stand by seed treatment was obtained only in one case with New Improved Ceresan at 1/2 oz. per bushel. There was no evidence of destruction of nodule bacteria by seed treatment, except that no nodules were found on the main root of plants from seed treated with organic mercurial fungicide. The bacteria causing bacterial pustule and bacterial blight were found to be widely distributed on the surface of beans from infected plants. Both organisms were destroyed at 10° F. in 7 weeks in culture solution, but withstood 13 weeks at the same temperature when dispersed in steamed soil.

234. Kernkamp, M. F. 1948. Should soybean seed be treated? Minnesota Farm & Home Sci. 5 (3): 5.
Seed treatment with Spergon followed by inoculation with nodule bacteria is recommended.

235. _____. 1948. Chemical treatment of soybean seed in relation to nodulation by nodule bacteria. Phytopath. 38: 955-959.
The application of Spergon with commercial nitragin did not influence nodulation by Rhizobium leguminosarum when soybean seeds were planted in soil that already contained the bacteria, but did reduce nodulation in steamed soil. The time of application of inoculant or fungicide did not appear to be important, although slightly more nodulation resulted when the inoculant was applied after the Spergon.

236. Kernkamp, M. F. 1948. Root rots of soybeans. Soybean Dig. 9 (11): 54-55.
Rhizoctonia solani is considered potentially the most destructive pathogen of soybean in Minnesota. Trials on its control are in progress along these lines, namely, crop rotation, seed treatment, and breeding.

237. Khan, A. M. 1950. Temperature and the development of Rhizoctonia solani on legumes. Phytopath. 40: 14.
The isolate from soybean killed more plants of soybean and pea at low (14° - 17° C.) and high (28° - 32°) temperatures than at intermediate temperatures. The isolate from flax behaved similarly as the soybean isolate on soybean, but its virulence on pea increased with temperature. The optimum temperatures for their growth in culture were 24° and 18° C., respectively, for the soybean isolate and flax isolate.

238. Kiesselbach, T. A. 1943. Crop response to hormone seed treatments. Jour. Amer. Soc. Agron. 35: 321-331.
No benefits were derived from hormone treatment of soybean seeds.

239. Kikuchi, R. 1924. On a disease of the soybean caused by Cercosporina. Utsunomiya Agr. Coll. Bull. 1: 1-19.
A purple spot disease, caused by Cercosporina sp., was found on soybean seeds in Japan. The morphology of the fungus is described.

240. King, T. H. 1948. Pod and stem blight of soybeans in Ohio, 1947. Plant Dis. Reptr. 32: 193.
A short note on the occurrence and spread of the disease.

241. Koehler, B. 1931. Diseases of soybeans in Illinois. Proc. Amer. Soybean Assoc. 3: 60-64.
An account, chiefly on symptoms and effects, of the following diseases: Mosaic, bacterial blight, pod and stem blight, Fusarium blight, root and stem rots caused by Pythium sp. and Sclerotium rolfsii, and downy mildew.

242. _____. 1943. Results of uniform seed treatment tests on soybeans. Plant Dis. Reptr. Suppl. 145: 76-79.
Yield increase was obtained by treating oil-type soybean with Semesan, Fermate, New Improved Ceresan, and Spergon, in half of the localities tested.

243. _____. 1944. New developments in soybean disease studies. Soybean Dig. 4 (8): 6-7.
The occurrence and prevalence of charcoal rot, pod and stem blight, bacterial pustule, and bud blight on soybean in U. S. A. are reviewed. Tobacco ring-spot virus causes spotting and falling off in Illinois, but causes bud blight in Iowa. In co-operative seed treatment tests, Spergon gave the highest stand increase in 3 northern States using a Manchu type of soybean. In the other 6 States, where the Lincoln variety was grown, the best results were obtained with Arasan. Only one case showed a significant increase of yield. In another series of tests, the emergence of Mukden seed type soybean was significantly benefited by seed treatment with Spergon and Arasan in 6 out of 13 States.

244. _____. 1946. Marked damage by brown stem rot. Soybean Dig. 7 (1): 17.
Soybean sustained heavy damage from the disease in 1946. The unnamed pathogen is soil-borne, but not carried on the seed.

245. Köhler, E. and K. Heinze. 1941. Übertragungsversuche mit dem Mosaikvirus der Sojabohne. Landw. Jahrb. 90: 233.
Soybean mosaic virus can be transmitted by rubbing of the sap. In bush bean and summer vetches the virus infection could be confirmed by re-transmission only from the leaves which have been rubbed. Of

the 6 species of aphids investigated, the transmission was most effective by Myzodes persicae and Macrosiphon solani, less certain by Doralis fabae, D. rhamni and D. frangulae. Transmission by Macrosiphon solanifolii is probable.

246. Kornfeld, A. 1933. Die Blattfleckenkrankheit der Soja -- eine Kalimangel-Erscheinung. Zeit. Pflanzenernähr. Dung. u. Bodenk. 32: 201-221.
In Rumania, the symptoms of potash deficiency include an irregular spotting and sometimes chlorosis of the foliage and precocious maturity with its concomitants of defoliation and reduction in yield. Among the internal modifications are starch accumulation and a feeble development of the blast of the stem with subsequent tendency to lodging. The affected plants are liable to attack by bacterial diseases.

247. _____. 1935. Schädigungen und Krankheiten der Ölbohne (Soja), soweit sie bisher in Europa bekannt geworden sind. Zeitschr. Pflanzenkr. 45: 577-613.
A detailed account on weeds, insect and other animal injuries, and physiological and parasitic diseases affecting soybeans in Europe. The diseases of physiological nature include those caused by acid soil, drought, deficiencies, frost, hail, and unsuitable harvesting and storing conditions. The parasitic diseases include: downy mildew, brown spot, frog-eye spot, Fusarium wilt, anthracnose, bacterial blight, and a rust-spotting disease caused Pseudomonas phaseoli. Two diseases of unknown nature, namely curly leaf and dwarfing, are also described.

248. Kovachevsky, I. C. 1938. (Parasitic fungi new for Bulgaria. 5th Contribution.) Rev. Inst. Rech. Agron. Bulgaria 8 (4): 3-13. (In Russian).
Includes Bacterium glycineum on soybean.

249. Kreitlow, K. W., and W. C. Price. 1948. A new virus disease of Ladino clover. Phytopath. 38: 15-16.
An abstract of the following entry.

250. _____, and _____. 1949. A new virus disease of Ladino clover. Phytopath. 39: 517-528.
A new virus disease of Ladino clover referred to as yellow patch can produce systemic infection on soybean by artificial inoculation.

251. Kreutzer, W. A. 1941. Host-parasite relationship in pink root of Allium cepa. III. The action of Phoma terrestris on Allium cepa and other hosts. Phytopath. 31: 907-915.
Phoma terrestris was isolated from the roots of soybean grown in infested soil, showing no external symptoms.

252. Kurata, H. 1950. (On the scab disease of soybeans). Byogaichu Hassei Yosatsu Shiro No. 24: 40-49. (In Japanese, mimeographed).
An hitherto unknown but destructive disease was found on soybean. In the field, leaves, stems and pods are affected. In severe cases of pod infection the seed may fail to develop. It is caused by Sphaceloma sp.

253. Kusano, S. 1932. The host-parasite relationship in Olpidium. Jour. Coll. Agr. Imp. Univ. Tokyo. 11: 359-426.
Olpidium trifolii and O. viciae can infect soybean through wounds by artificial inoculation.

254. Kvashnina, E. S. 1928. (Preliminary report of the survey of diseases of medicinal and industrial plants in North Caucasus.) Bull. N. Caucasian Pl. Prot. Sta. 4: 30-46. (In Russian)
Soybean suffered from two undetermined bacterial diseases, one on leaves and the other on leaves and pods.

255. Kvičala, B. 1937. (Susceptibility of soybean varieties to bacterial blight, as determined by artificial inoculations. Preliminary communication.) (Ann. Acad. Tchécosl. Agr.)

12: 266-271. (In Czechoslovakian with German summary).
Among eight soybean varieties tested in Czechoslovakia, Bratislavska yellow Sl. 1, Plattska large yellow, and Brnenska Chmelarova SVA 1 were most resistant to bacterial blight.

256. Larsh, H. W. 1944. Soybean diseases in Arkansas. Plant Dis. Reptr. 28: 870-871.
Soybeans were infected by bacterial blight, bacterial pustule, frog-eye spot, Alternaria leaf spot, Fusarium blight, downy mildew, charcoal rot, southern blight, and mosaic.

257. _____. 1944. Diseases observed on soybeans in Arkansas. Plant Dis. Reptr. 28: 956-957.
Records the prevalence of the following diseases: Bacterial pustule, wildfire, pod and stem blight, anthracnose, downy mildew, frog-eye spot, charcoal rot, and mosaic.

258. _____. 1944. Diseases reported on soybean in Oklahoma. Plant Dis. Reptr. 28: 1010.
Records the prevalence of bacterial pustule, wildfire, pod and stem blight, frog-eye, charcoal rot, yeast spot and mosaic.

259. _____. 1944. Diseases observed on soybeans in Arkansas. Plant Dis. Reptr. 28: 1125-1126.
Records the prevalence of bacterial pustule, anthracnose, frog-eye, wildfire, stem blight, and charcoal rot.

260. _____. 1944. Summary report of plant diseases in Oklahoma, 1943. Plant Dis. Reptr. Suppl. 149: 317-326.
Reporting frog-eye leaf spot, pod and stem blight, anthracnose, wilt caused by Fusarium oxysporum f. tracheiphilium (F. bulbigenum var. tracheiphilum), Nematospora coryli, charcoal rot, bacterial blight, bacterial pustule, and mosaic.

261. Le Beau, F. J. 1947. A virus-induced top necrosis in beans. Phytopath. 37: 434.
A virus which causes severe systemic necrosis of beans can infect soybeans by artificial inoculation. Its thermal inactivation point suggests a relationship with that responsible for soybean bud blight.

262. Lehman, S. G. 1922. Pod and stem blight of soybean. Jour. Elisha Mitchell Sci. Soc. 38: 13.
Phomopsis sojae n. sp. was isolated from the stems, pods, and seeds, and has been observed to cause the death of young soybean plants. It overwinters in diseased stem and seed. The ploughing under of the diseased plants, use of healthy seeds, and crop rotation are recommended..

263. _____. 1923. Pod and stem blight of soybean. Ann. Missouri Bot. Gard. 10: 119-169.
The perfect stage of Phomopsis sojae was developed in culture and named Diaporthe sojae. Full description is given to the disease and the fungus. The infection and dissemination are favored by high humidity. The pycnospores germinated best at pH 4.1-6.1. Light was essential to the formation of pycnidia. Black Eyebrow was the most susceptible variety of soybean to the disease.

264. _____. 1928. Frog-eye leaf spot of soybean caused by Cercospora diazu Miura. Jour. Agr. Res. 36: 811-833.
The symptoms, and the morphology and cultural characters of the fungus are described. The fungus injures the host by means of some substance acting in advance of the hyphae as evidenced by the alteration in the staining reaction of the host cells. It overwinters on diseased stems, leaves, and seeds. Control measures include ploughing under of the crop after harvest, rotation, and the use of early maturing varieties.

265. Lehman, S. G. 1929. Studies on bacterial pustule of soybean. Phytopath. 19: 96. An abstract of the following entry.

266. _____. 1931. Observations and experiments relating to the bacterial pustule disease of soybean. Jour. Elisha Mitchell Sci. Soc. 46: 179-189.
Bacterium phaseoli var. sojense was isolated from lesions on soybean showing no sign of pustular development, which was believed not a necessary concomitant of the infection. The infection was heavier on plants kept at a constant temperature of 30°-33° C. than on those exposed to fluctuations between 22° and 30° C. The fungus was able to survive on dry leaves from one season to another.

267. _____. 1931. Powdery mildew of soybean. Jour. Elisha Mitchell Sci. Soc. 46: 190-195.
The causal fungus was identified as Erysiphe polygoni, but the strain from garden bean was unable to infect soybeans.

268. _____. 1934. Frog-eye (Cercospora daizu Miura) on stems, pods, and seeds of soybean, and the relation of these infections to recurrence of the disease. Jour. Agr. Res. 48: 131-147.
The fungus was frequently observed on the stems, pods, and seeds of soybeans. In the stems, the parasite is chiefly confined to the cortex and the injury to the phloem and cambium is usually due to the diffusion of the toxic substances from the necrotic cortex. In the pods, the mycelium penetrates through the pod wall, entering the thin white membranes lining the pod and closely investing the seed. The growth of the fungus is usually superficial on the seeds and is easily controlled by seed disinfectants. The infection carries over winter on diseased leaves and stems, but ploughing in of infected stubbles is not practicable in preventing the disease. Further spread of the disease may be prevented by the use of healthy seeds.

269. _____. 1942. Notes on plant diseases in North Carolina in 1941: Soybeans. Plant Dis. Reptr. 26: 111.
Records the prevalence of bacterial pustule, frog-eye, and downy mildew.

270. _____. 1943. Occurrence of yeast spot on soybeans in North Carolina. Plant Dis. Reptr. 27: 602.
Nematospora phaseoli ? or N. coryli ? was isolated from soybean seeds showing yellowish to brown areas.

271. _____. 1944. Dusting soybean for control of bacterial pustule. Phytopath. 34: 1007-1008.
Copper dusts were effective in reducing the disease, but sulfur dust was not.

272. _____. 1945. Three important foliage diseases of soybeans. Res. & Farming North Carolina Agr. Exp. Sta. 4 (1): 4-5.
A popular account of bacterial blight, bacterial pustule, and brown spot. Reaction of soybean varieties grown in North Carolina to bacterial pustule is given.

273. _____. 1946. Control of bacterial pustule of soybean by dusting. Phytopath. 36: 405.
Among materials tested, only copper dusts reduced the disease.

274. _____. 1947. Powdery mildew of soybean. Phytopath. 37: 434.
Describes briefly Microsphaera sp. on soybean found in North Carolina. Varietal reaction to the disease was noted.

275. Lehman, S. G. 1950. Purple stain of soybean seeds. North Carolina Agr. Exp. Sta. Bull. 369.
The disease has become increasingly prevalent since 1927. The fungus survives in infected seeds and spreads by wind. It is chiefly confined to the seed coat. Seedlings from infected seeds are stunted or killed after emergence. The disease appears on a high proportion of seeds when the crop matures under rainy conditions. Varieties differ in susceptibility. Dusting plants with a mixture containing 7% copper gave partial control.

276. _____, and J. H. Graham. 1948. Results from dusting soybeans with copper in 1947. Phytopath. 38: 570.
Dusting soybean with a mixture consisting of tribasic copper, wheat flour, DDT and Cherokee clay reduced a severe leaf disease of unidentified cause and increased the yield significantly.

277. _____, and _____. 1948. Soybean seed treatment tests in North Carolina in 1947. Phytopath. 38: 571.
Emergence was increased and diseased seedlings decreased more by Arasan than by Spergon.

278. _____, and R. F. Poole. 1929. Research in botany. North Carolina Agr. Exp. Sta. Ann. Rept. 51 (1928): 59-67.
Cercospora daizu may remain alive on moist diseased leaves until next planting season, but is unable to survive the disintegration of the leaves. Seed treatment proved unavailing. Good control of downy mildew was obtained by seed treatment. The bacterial pustule organism was found to remain viable in dry diseased leaves as well as partially decayed leaves left out-of-doors from one season to another. Columbia variety of soybean was practically immune.

279. _____, and F. A. Wolf. 1924. A new downy mildew of soybeans. Jour. Elisha Mitchell Sci. Soc. 39: 164-169.
The fungus is named Peronospora sojae.

280. _____, and _____. 1926. Pythium root rot of soybean. Jour. Agr. Res. 33: 375-380.
The symptoms of the disease and the morphology of Pythium debaryanum are described.

281. _____, and _____. 1926. Soybean anthracnose. Jour. Agr. Res. 33: 381-390.
The ascogenous stage of Colletotrichum glycines was found on diseased stem of soybean and also in culture. It differs from Glomerella cingulata in morphology and was named G. glycines.

282. _____, and J. W. Woodside. 1929. Varietal resistance of soybean to the bacterial pustule disease. Jour. Agr. Res. 39: 795-805.
Fifty-five soybean varieties are listed showing their reaction to the disease. Columbia variety possesses greatest resistance. Other highly resistant varieties include Mandarin and Dominion.

283. Lepik, E. 1938. Phytopathologische Notizen 10. Bull. Phytopath. Exp. Sta. Univ. Tartu. 43, pp. 213-225.
Records Phyllosticta sojaecola on soybean in Estonia.

284. Lihnell, D. 1939. Några iakttagelser rorande sjukdomar på soja i vart land. Vaxtskyddsnotiser, Växtkyddsanst., Stockh., 3(4-5): 69-73.
An account of symptoms and effects of the following diseases: Bacterial blight, downy mildew, Sclerotinia stem rot, and mosaic.

285. Likhite, V. N. 1936. Host range of the Gujarat cotton root rot. Proc. Ass'n. Econ. Biol., Coimbatore, 3: 18-20.
Macrophomina phaseoli infected soybeans in the field tests in India.

286. Ling, L. 1940. Seedling stem blight of soybean caused by Glomerella glycines. Phytopath. 30: 345-347.
Seedling blight occurs before or after emergence. The mycelium that survives in the infected seed and the infested soil serves as the primary source of inoculum.

287. _____. 1948. Host index of the parasitic fungi of Szechwan, China. Plant Dis. Reptr. Suppl. 173: 1-38.
Includes 20 fungi parasitic to soybean.

288. _____, and J. Y. Yang. 1944. Studies on the biology and pathogenicity of Colletotrichum indicum. Ann. Bot., (London) n. s. 8: 91-104.
Soybean pods were infected by the fungus in inoculation tests.

289. Link, G. K. K., and C. G. Sharp. 1927. Correlation of host and serological specificity of Bacterium campestris, Bact. flaccumfaciens, Bact. phaseoli and Bact. phaseoli sojense. Bot. Gaz. 83: 145-160.
Agglutination tests show that the biological specificity displayed by these bacteria in their host relations is associated with serological specificity.

290. Liu, K. 1940. Studies on a Fusarium disease of soybean pods. Mem. Coll. Agr. Kyoto Imp. Univ. 47: 15-29.
The fungus causing a destructive pod blight is morphologically identical to Fusarium bulbigenum var. tracheiphilum. Under natural conditions the pathogen is confined to pods, but infects the stem by artificial inoculation. Conidia germinate at temperatures from 16° to 36° C., the optimum temperature being 24° C.

291. Liu, S. T. 1948. Seed-borne diseases of soybean. Bot. Bull. Acad. Sinica 2: 69-80.
The following fungi were isolated from soybean seeds in China: Diaporthe phaseolorum var. sojae, Cercospora kikuchii, C. vignicola, Colletotrichum glycines, Gloeosporium sp., Phyllosticta sojaecola, Alternaria atrans, Macrophoma mame, Diplodia sp., Mycosphaerella sojae, Rhizoctonia solani, and Helminthosporium sp.

292. Lobik, V. I. 1930. (The problem of the diseases of the soybean in the light of observations in 1930 at Essentuki.) Bull. North Caucasian Pl. Prot. Sta., Rostoff-on-Don, 6-7, p. 285. (In Russian).
Soybeans were attacked by the following diseases: Bacterial leaf spots, wilt caused by Fusarium sp., downy mildew, and Botrytis sp.

293. Loukyanovitch, F. K., L. A. Lebedeva, V. A. Kizeritsky, O. I. Ermolayeva, and S. I. Obolensky. 1931. (Pests and diseases of agricultural crops in the region of the Turkestan-Siberian Railway) Pl. Prot., Leningrad, 7: 349-360. (In Russian).
In western Siberia, the following diseases were present on soybean: Bacterium sojae, Fusarium sp. causing seedling blight, downy mildew, Sclerotinia sclerotiorum, and Ascochyta sp. on pods.

294. Lowig, Emil. 1936. Kalimangelerscheinungen bei der Sojabohne. Ernähr. Pflanze 32: 113-114.
Illustrations showing different stages of potash starvation.

295. Luttrell, E. S. 1945. Additional hosts of Diaporthe sojae. Plant Dis. Reptr. 29: 89-90.
The fungus was observed chiefly on weakened or senescent plants of soybean and other crops, and was almost constantly associated with other fungi, notably Macrophomina phaseoli.

296. Luttrell, E. S. 1947. Diaporthe phaseolorum var. sojae on crop plants. Phytopath. 37: 445-465.
The fungus was proved to be merely a vigorous saprophyte that may fruit quickly upon dead plants. It differs from Diaporthe phaseolorum, in pathogenicity, in the production of pycnidia in culture, and in the morphology of spores and stromata. It has been found on 12 hosts other than soybean.

297. Major, T. G. 1925. Report on tobacco disease investigations. Rept. Tob. Div. Dom. Exp. Farms, Canada, 1923, pp. 38-41.
Thielavia basicola can infect soybeans by artificial inoculation.

298. Malloch, W. S. 1923. The problem of breeding nematode resistant plants. Phytopath. 13: 436-450.
Soybean was found to be infected by Heterodera radicicola.

299. Manns, T. F. 1913. A bacterial disease of the sweet pea and clovers. Phytopath. 3: 74-75.
Bacillus lathyri is pathogenic to soybeans.

300. _____, and J. F. Adams. 1924. Department of Plant Pathology and Soil Bacteriology. Delaware Agr. Exp. Sta. Ann. Rept. 1923, pp. 25-48.
Mottling and atrophy of leaves of soybean were caused by a mosaic disease.

301. _____, and _____. 1925. Department of Plant Pathology. Delaware Agr. Exp. Sta. Ann. Rept. 1925. (Bull. 141: 24-30.)
Records the following diseases on soybeans: Leaf spot caused probably by Septoria glycines, downy mildew, and bacterial pustule.

302. _____, and _____. 1929. Department of Plant Pathology. Delaware Agr. Exp. Sta. Ann. Rept. 1929 (Bull. 162: 53-67).
Brown spot, downy mildew, bacterial pustule, and anthracnose were found in soybean seed disinfection plots. Seed disinfection failed to control the seed-borne foliage diseases.

303. _____, and _____. 1934. Department of Plant Pathology. Delaware Agr. Exp. Sta. Ann. Rept. 1933 (Bull. 188: 36-46).
Includes notes on the prevalence of bacterial pustule.

304. Marchionatto, J. B. 1947. Hongos parasitos de las plantas, nuevos o poco conocidos en la Argentina. Publ. Misc. Minist. Agr. Buenos Aires, Ser. A., iii, 37. 11 pp.
Sclerotinia sclerotiorum was isolated from soybeans.

305. Martin, J. N. 1942. The effect of freezing temperatures in December 1941 and January 1942 in Story, Boone and Polk counties on the viability of soybeans of the 1941 harvest. Proc. Iowa Acad. Sci. 49: 215-216.
Most of the yellow soybeans in the farmers' bins in central Iowa endured the fall and January cold without considerable impairment of viability, whereas those beans remaining in the field were damaged beyond any use for seed.

306. Martin, J. P. 1944. Pathology. Hawaii Sug. Exp. Sta. Rept. 1942-1943. pp. 19-28.
Pseudomonas glycines on soybean was under investigation.

307. Martyn, E. B. 1944. Plant Pathological Division. Dept. Agr. Jamaica Rept. 1942-43, p. 16.
Glomerella glycines caused damage to soybean pods ripening in wet weather.

308. Massalongo, C. 1900. De nonnullis speciebus novis micromycetum agri veronensis. Atti R. Inst. Veneto Sci. Lett. ed Arti. 59: 683-690.
Includes a description of Phyllosticta sojaecola n. sp.

309. Matsumoto, T. 1928. Beobachtungen über Sporenbildungen des Pilzes Cercosporina kikuchii. Ann. Phytopath. Soc. Japan 2: 65-69.
Temperatures between 15° to 20° C. and potato dextrose agar were found to be most suitable for the production of conidia in culture.

310. _____, and R. Tomoyasu. 1925. Studies on purple speck of soybean seed. Ann. Phytopath. Soc. Japan, 1: 1-14.
The symptoms and the morphology of the causal fungus, Cercosporina kikuchii, are described. The fungus turns glucose media purple and its mycelial extract in water contains a purple pigment which is insoluble in a number of solvents.

311. McLaughlin, J. H. 1942. Notes on diseases of soybean and other legumes in Oklahoma. Plant Dis. Reptr. 26: 356-359.
Among soybean varieties observed, only Chief, Arksoy 152, C-146, Ogden, Habaro and Sciota remained relatively unaffected by bacterial pustule.

312. _____. 1946. Vegetable seed treatment for Oklahoma. Oklahoma Agr. Exp. Sta. Bull. 293.
Damping-off of soybean was caused by Pythium debaryanum and Rhizoctonia solani. Significant increase of stand was obtained by treating seeds with Spergon.

313. McMurtrey, J. E., Jr. 1948. Visual symptoms of malnutrition in plants. In "Diagnostic techniques for soils and crops". pp. 231-289.
In the appendix includes brief summaries on symptoms of malnutrition of soybean due to deficiencies of boron, calcium, iron, magnesium, nitrogen, phosphorus, potassium, and sulfur.

314. McNew, G. L. 1948. Study of soybean diseases and their control. Iowa Agr. Exp. Sta. Rept. on Agricultural Research for the year ending June 30, 1948. pp. 188-189.
Five varieties of soybean tested were equally susceptible to Colletotrichum. Seed treatment partially controlled hypocotyl infection by Pythium. Alternaria sp. causing spots on pods and leaves and Cercosporina sp. causing purple speck of seed were isolated.

315. McRae, W. 1933. Report of the Imperial Mycologist. Imp. Inst. Agr. Res. Sci. Rept. 1931-1932, pp. 122-140.
Cercospora dolichi infected soybeans in inoculation tests.

316. Meehan, Frances, and H. C. Murphy. 1946. A new Helminthosporium blight of oats. Science. 104: 412-414.
The fungus also occurs as a weak parasite on soybean.

317. Melhus, I. E. 1942. Soybean diseases in Iowa in 1942. Plant Dis. Reptr. 26: 431-432.
Records the occurrence and prevalence of bacterial blight, mosaic, downy mildew, anthracnose, pod and stem blight, and bud blight.

318. _____, J. N. Martin, and H. C. Murphy. 1944. The influence of pythiaceous and other fungi on seedling stands of legumes and other crops. Iowa Agr. Exp. Sta. Rept. on Agricultural Research for the year ending June 30, 1949. Part 1, p. 157.
Seed treatment of soybean increased the stands with some materials, but not the yield. Pythium debaryanum, Rhizoctonia sp. and Fusarium sp. were the organisms most frequently obtained from the roots. Colletotrichum glycines, Diaporthe sojae, Pythium spp. and a soft rot bacterium were isolated from rotting seeds.

319. Miller, P. R., and N. W. Nance. 1949. Preliminary estimates of acreages of crop lands in the United States infested with some organisms causing plant diseases. Plant Dis. Reptr. Suppl. 185: 207-252.

Rhizoctonia was of minor importance in Minnesota on soybean, but affected 10,000 acres in Oklahoma. Sclerotium bataticola affected 60 percent of soybean acreage in Kansas.

320. Miller, P. R., and Jessie I. Wood. 1947. An evaluation of certain phases of the Emergency Plant Disease Project. Plant Dis. Reptr. Suppl. 167: 1-26.
During the Emergency Plant Disease Prevention Project surveys, seven new diseases were found on soybean, caused by Helminthosporium vignae, Microsphaera sp., Myrothecium roridum, Nematospora coryli, Penicillium sp., Phyllosticta sojaecola, and Pseudomonas tabaci. Another disease, bud blight, was found in States where it had not previously been known to occur.

321. Milner, M., and W. F. Geddes. 1946. Grain storage studies. IV. Biological and chemical factors involved in the spontaneous heating of soybeans. Cereal Chem. 23: 449-470.
Initial temperatures rise to between 50° and 55° C. and parallel respiratory increase were directly associated with the multiplication of Aspergillus glaucus and A. flavus. Surface sterilization of the seeds failed to eliminate mould infection, but inoculation of autoclaved soybeans with A. flavus spores resulted in heating and respiration curves virtually identical with those of original sample.

322. Mitra, M. 1936. Report of the Imperial Mycologist. Agr. Res. Inst., Pusa, Sci. Rep. 1933-1934, pp. 139-167.
A Cercospora found on leaf, stem, and pod of soybeans in India does not agree with C. daizu.

323. Miura, M. 1930. Diseases of the main agricultural crops in Manchuria. Agr. Exp. Sta. Manchuria Railw. Co. Bull. 11, rev. ed., 56 pp. (In Japanese).
The symptoms, causes, and control measures are given for the following diseases: Bacterium sojae var. japonicum, Peronospora manshurica, Sclerotinia libertiana, Gibberella sp., Hypochnus centrifugus, Uromyces sojae, Septoria glycines, Cercospora sojina, Cercosporina kikuchii, Heterodera schachtii, Pleosphaerulina sojaecola, and Cuscuta chinensis. Other diseases not occurring in Manchuria are also noted.

324. Miyagi, J. 1936. Studies on purple and brown spots on soybean seeds. Proc. Crop. Sci. Soc. Japan. 8: 65-82. (In Japanese).
Gives no organism but states that some authors consider Fusarium sp. or Cercosporina kikuchii as the cause.

325. Moore, E. S. 1930. Internal boll disease of cotton in South Africa. S. Afr. Dept. Agr. Sci. Bull. 94: 11-18.
Soybean seeds were infected by Nematospora coryli and N. gossypii.

326. Morse, W. J., and J. L. Carter. 1939. Soybeans: culture and varieties. U. S. Dept. Agr. Farmers' Bull. 1520.
A popular account including discussions on the following diseases: Purple spot of seeds, bacterial blight, bacterial pustule, mosaic, wilt, brown spot, sunburn or aphid injury, downy mildew, pod and stem blight, anthracnose, sclerotial stem rot, frog-eye spots, and Pythium root rot.

327. Muller, A. S. 1934. Brazil: Preliminary list of diseases of plants in the State of Minas Geraes. Intern. Bull. Pl. Prot. 8: 193-198.
Includes Bacterium sojae on soybean.

328. _____. 1936. Brazil: Some new records of plant diseases in the State of Minas Geraes. Intern. Bull. Pl. Prot. 10: 98-99.
Includes Cercospora stevensii on soybean.

329. Murata, T. 1925. (Soybean mosaic.) Jour. Pl. Prot. (Tokyo) 12: 451-452. (In Japanese).
A review of entry 134.

330. Nacion, C. C. 1924. Study of Rhizoctonia blight of beans. Philipp. Agr. 12: 315-321.
Inoculation of soybean with Rhizoctonia solani isolated from lima bean resulted in typical Rhizoctonia symptoms.

331. Naftel, J. A. 1937. Soil-liming investigations: V. The relation of boron deficiency to over-liming injury. Jour. Amer. Soc. Agron. 29: 761-771.
Over-liming in the light-textured loamy sandy soil in Alabama resulted in injury to soybean and other crops. The symptoms are typical of boron deficiency. The application of phosphorus, manganese, or calcium silicate did not overcome the injurious effect, but the incorporation of micro-elements including 1 p. p. m. of boron completely prevented the damage.

332. Nagorny, P. O., and E. M. Eristavi. 1929. (A brief survey of plant diseases in Abkhasia in 1928.) Agr. Exp. Sta. Abkhasia Publ. 38: 1-38. (In Russian).
Includes a brief description of Phyllosticta sojaecola on soybean.

333. Nakano, K. 1919. (Leaf scorch of soybean). Jour. Pl. Prot. (Tokyo). 6: 217-221. (In Japanese).
An account of the symptoms and the causal organism which is named Pseudomonas glycines n. sp.

334. Nakata, K. 1940. (Report of diseases of agricultural and horticultural crops in North China and Mongolia.) Agr. Exp. Sta. North China Surv. Rept. 1: 1-92. (In Japanese).
Includes the following diseases on soybean: Downy mildew, bacterial pustule, Phyllosticta sp., and mosaic.

335. _____, and H. Asuyama. (Report on diseases of main agricultural crops in Manchuria.) Bureau Indus. Manchuria Rept. 32: 1-166. (In Japanese).
Includes the following diseases on soybean: Bacterial pustule, downy mildew, chlorosis, mosaic, Phyllosticta sojaecola, Cercospora sojina, Cercosporina kikuchii, and wilt of unknown cause.

336. _____, and K. Takimoto. 1934. (A list of crop diseases in Korea.) Agr. Exp. Sta. Govn.-Central Chosen Res. Rept. 15: 1-146. (In Japanese).
Includes the following diseases on soybean: Bacterium sp. causing leaf-spot, Mycosphaerella sojae, downy mildew, Phyllosticta sojaecola, Septoria glycines, Cercospora sp., Hypochnus centrifugus, Cercosporina kikuchii, Colletotrichum glycines, and Gloeosporium sp.

337. Nance, Nellie W. 1948. Some unusual or outstanding plant disease developments in the United States in 1947. Plant Dis. Reptr. Suppl. 177: 143-169.
Occurrence of pod and stem blight in Ohio, and of Phyllosticta glycineum in Missouri.

338. _____. 1950. Some new or noteworthy plant disease records and outstanding developments in the United States in 1949. Plant Dis. Reptr. Suppl. 194: 364-380.
Referring to entry 102.

339. Naoumoff, N. 1914. Materiaux pour la flore mycologique de la Russie, Fungi Ussurienses I. Bull. Soc. Myc. France. 30: 64-83.
Contains a technical description of Peronospora trifoliorum var. manshurica n. var.

340. Narasimhan, M. J. 1934. Report of work done in the Mycological Section during 1932-1933. Agr. Dept. Mysore, Admin. Rept. 1932-1933, pp. 53-56.
Rhizoctonia sp. occurred on soybean.

341. Nelson, L. B., and W. H. Allaway. 1948. Yellow soybeans may require iron. What's New in Crops and Soils (Dec.).
Iron-deficient soybeans were found in Iowa on a calcareous soil with a pH of 7.9.

342. Nelson, W. L., and F. E. Bear. 1949. Plant-nutrient deficiency symptoms in legumes. In Hunger Signs in Crops. 2nd Ed. pp. 269-296. Amer. Soc. Agron. & Nat. Fert. Ass'n., Washington.
Includes descriptions with illustrations on the symptoms of nutritional disorders of soybean due to boron toxicity, and deficiencies of iron, magnesium, manganese, nitrogen and potassium.

343. Nisikata, T. 1938. Studies on the brown mottling of soybean. Res. Bull. Agr. Exp. Sta. S. Manchuria Railway Co. 24: 83-94.
In comparison with healthy seed, the mottled seed contained less crude fat, more crude protein, and less sugars, but the amount of reducing sugar was increased in the endosperm of mottled seed. Among the inorganic matters in seed coat, Mn_3O_4 and Fe_2O_3 + Al_2O_3 were increased significantly by mottling.

344. _____. 1938. Studies on brown mottling of soybean. II. Several considerations on the factors producing the brown mottling. Res. Bull. Agr. Exp. Sta. S. Manchuria Railway Co. 25: 43-59.
The moisture content of soil is considered as an important factor in producing mottling. The application of iron and manganese into soil significantly increased the mottled seed.

345. Noble, R. J. 1931. New South Wales: Plant diseases. Intern. Bull. Pl. Prot. 5: 202-205.
Bacterial blight and bacterial pustule occurred on soybeans.

346. _____. 1937. Australia: Notes on plant diseases recorded in New South Wales for the year ending 30th June, 1937. Intern. Bull. Pl. Prot. 11: 246-247.
A witches' broom-like condition occurred on soybeans.

347. _____, H. J. Hynes, F. C. McCleery, and W. A. Birmingham. 1935. Plant diseases recorded in New South Wales. Dept. Agr. New South Wales Sci. Bull. 46.
Lists Alternaria sp. and Phyllosticta sp. causing leaf spots, Bacterium sp. causing blight, Bacterium phaseoli sojense, Fusarium sp. causing root rot, Heterodera marioni, and Sclerotium rolfsii on soybeans.

348. _____, _____, _____, and _____. 1937. Plant diseases recorded in New South Wales. Supplement No. 1. 7 pp. New South Wales Dept. Agr.
Lists Bacterium glycineum on soybean.

349. Nojima, T. 1926. (On a disease of soybean pods due to the parasitism of a Fusarium fungus. Preliminary report. Jour. Pl. Prot. (Tokyo) 13: 138-147. (In Japanese).
A species of Fusarium with white to light pinkish mycelium produced numerous salmon-pink spots on the pods of soybean. Infection occurred chiefly through wounds. The fungus was unable to attack the roots, leaves and stems.

350. Noll, W. 1939. Untersuchungen über Fuss- und Welkekrankheiten bei Leguminosen. Zeit. Pflanzkh. 49: 385-431.
Three strains of Ascochyta pinodella isolated from peas, broad beans, and soybeans were pathogenic to the same three hosts in Germany.

351. Okabe, N. 1932. Bacterial diseases of plants occurring in Formosa I. Jour. Soc. Trop Agr. 4: 470-483.

The symptoms of bacterial pustule of soybean are described. According to the morphology and physiology, the causal organism is probably Bacterium phaseoli var. sojense, though a close relationship with B. phaseoli and B. glycines is also indicated.

352. Okabe, N. 1933. Bacterial diseases of plants occurring in Formosa III. Jour. Soc. Trop. Agr. 5: 157-166.
Includes a full description of the symptoms of bacterial leaf spot and its pathogen, Bacterium sojae var. japonicum. The minimum, optimum, and maximum temperatures for the growth of the pathogen are 3°, 25°, and 37° C. respectively, with the thermal death point at 47° C.

353. Olive, L. S. 1949. Target spot of cowpea and soybean. Mycologia 41: 355.
Proposes Helminthosporium vignicola (Kawamura) n. comb., with Cercospora vignicola Kawam. and H. vignae Olive as its synonyms.

354. _____, D. C. Bain, and C. L. Lefebvre. 1945. A leaf spot of cowpea and soybean caused by an undescribed species of Helminthosporium. Phytopath. 35: 822-831.
Helminthosporium vignae n. sp. was found to cause a small brown leaf spot on soybean. The soybean race differs from the cowpea race in the mildness in infectivity.

355. _____, and E. A. Walker. 1944. A severe leaf spot of soybean caused by Phyllosticta sojaecola. Plant Dis. Reptr. 28: 1122-1123.
A brief note on the occurrence in Ontario and Maryland of Phyllosticta leaf spot.

356. Owen, F. V. 1927. Hereditary and environmental factors that produce mottling in soybeans. Jour. Agr. Res. 34: 559-587.
All varieties with yellow or green seed tested proved to be subject to mottling. Both hereditary and environmental factors were important.

357. Pady, S. M. 1944. Bacterial pustule in Kansas. Plant Dis. Reptr. 28: 835.
Bacterial pustule was the only disease found on soybean.

358. _____. 1944. Notes on diseases observed in the Nebraska plant disease survey, August to November, 1943. Plant Dis. Reptr. Suppl. 149: 311-313.
Occurrence of bacterial pustule, pod and stem spot of unknown cause, and mosaic on soybean.

359. _____. 1944. Notes on the plant disease survey in Kansas, August to November, 1943. Plant Dis. Reptr. Suppl. 149: 313-316.
Reporting bacterial pustule, pod and stem spot of unknown cause, pod and stem blight, anthracnose, charcoal rot, and mosaic on soybean.

360. Pape, H. 1921. Pilzliche Schädlinge der Sojabohne. Mitteil. Biol. Reichsanst. Land. u. Forstw. 21: 36-42.
Two diseases caused by Sclerotinia libertiana and Botrytis cinerea are fully described. Both attack pods as well as growing plants.

361. _____. 1921. Beobachtungen bei Erkrankungen durch Botrytis. Gartenflora 70: 48-50.
Botrytis cinerea is able to attack first the withering young pods on the upper part of fully grown soybean plants and then proceed to the main axis.

362. Park, M. 1941. Report on the work of the Division of Plant Pathology. Acting Dir. Agr. Ceylon, Admin. Rept. 1939, pp. D20-D22.
Occurrence of Uromyces sojae ? in Ceylon.

363. Parris, G. K. 1940. A check list of fungi, bacteria, nematodes and viruses occurring in Hawaii and their hosts. Plant Dis. Reptr. Suppl. 121: 1-91.
Lists Heterodera marioni and Pratylenchus pratensis under soybean.

364. Paul, W. R. C. 1939. Report on the work of the Division of Plant Pathology. Dir. Agr. Ceylon, Admin. Rept. 1938, pp. D41-D45.
A stem disease caused by Rhizoctonia bataticola occurred on soybean.

365. Person, L. H. 1944. Parasitism of Rhizoctonia solani on beans. Phytopath. 34: 1056-1068.
Isolates of Rhizoctonia solani are classified into four groups according to their parasitism. Group 1 consisting of isolates from sugarcane and potatoes is non-pathogenic to soybeans, and group 3 consisting of rice isolate is weakly pathogenic to soybeans. Group 2 consisting of pea isolates and group 4 consisting of isolates from beans, tomato, eggplant and sugarbeet are pathogenic to soybeans, but can be further differentiated on soybeans and other legumes.

366. _____. 1944. List of plant diseases observed during surveys in Mississippi and Louisiana, August to November, 1943. Plant Dis. Reptr. Suppl. 148: 280-283.
Occurrence of Cercospora sojina, Diaporthe sojae, Sclerotium bataticloa, S. rolfsii, Pseudomonas glycinea, Xanthomonas phaseoli var. sojense, and mosaic.

367. Pethybridge, G. H. 1926. Fungus and allied diseases of crops. 1922-1924. Min. Agr. Misc. Publ. 52: 1-97.
Bacillus lathyri was isolated from soybeans.

368. Petty, M. A. 1943. Soybean disease incidence in Maryland in 1942 and 1943. Plant Dis. Reptr. 27: 347-349.
The following diseases are briefly noted: Downy mildew, purple seed spot probably caused by Cercospora kikuchii, bacterial blight, bacterial pustule, stem canker caused by Diaporthe sojae, frog-eye spot, leaf spot and stem blight probably caused by Septoria glycines, Alternaria sp. causing leaf spot, wilt caused by Fusarium spp., Rhizoctonia damping-off, charcoal rot, mosaic, potash deficiency, and sunscald.

369. _____. 1944. Soybean diseases on the Eastern Shore of Maryland. Bull. Sta. Board Agr. Delaware. 33 (5): 58-62.
Seed samples of 1943 crop showed injury from purple seed spot and white incrustations of downy mildews. In the fields, bacterial pustule, downy mildew, charcoal rot, frog-eye, and mosaic were prevalent. Results of seed treatment were inconclusive.

370. Pierce, W. H. 1934. Viroses of the bean. Phytopath. 24: 87-115.
By artificial inoculation, yellow bean mosaic virus (Pea virus 2) and lucerne mosaic virus (lucerne virus 2) can infect soybean.

371. _____. 1935. The identification of certain viruses affecting leguminous plants. Jour. Agr. Res. 51: 1017-1039.
Soybean mosaic differs from other viruses in the host range specific to soybean. Enation pea mosaic (pea virus 1) also can infect soybean in inoculation tests.

372. Pinckard, J. A. 1942. Diseases of soybeans and peanuts in Mississippi. Plant Dis. Reptr. 26: 472-473.
Records the occurrence and prevalence of bacterial pustule, downy mildew, mosaic, and Sclerotium blight.

373. Piper, C. V., and W. J. Morse. 1923. The soybean. 329 pp. McGraw-Hill.
Including a review of literature on soybean diseases published before 1921.

374. Porter, R. H. 1944. Soybean seed treatments. Plant Dis. Reptr. Suppl. 145: 22-25.

In 1943 co-operative tests, emergence was increased in certain localities by seed treatment, but yield was not affected in all cases.

375. Porter, R. H. 1946. Induced baldhead in soybean. Phytopath. 36: 168-170.
Soybean seeds planted in soils with a 15 percent moisture content and naturally infested by Pythium graminicola and P. debaryanum produced baldhead seedlings in which the plumule is partially or wholly destroyed. Seed protectants reduced the infection.

376. _____. 1946. Vegetable soybean seed treatments. Plant Dis. Reptr. Suppl. 161: 42-44.
Results of tests in 1944 using Banzei soybean with a high vitality show significant increase in emergence from treated seeds in 5 out of 17 locations. Spergon gave the most consistent increase.

377. _____. 1946. Germinability of treated and untreated lots of vegetable seed in Pythium-infested soil and in the field. Iowa Agr. Exp. Sta. Res. Bull. 345.
Arasan, Spergon, and Semesan Jr. were effective as protectants for soybeans against Pythium spp.

378. Porter, R. P., and H. T. Cook. 1942. Peronospora on soybeans in Virginia. Plant Dis. Reptr. 26: 413.
The downy mildew was abnormally abundant in 1942, because of the abundant rainfall and moderate temperature during the latter part of August.

379. Presley, J. T., and W. B. Allington. 1947. Brown stem rot of soybean caused by a Cephalosporium. Phytopath. 37: 681-682.
A brief note on the morphology and cultural characters of the fungus.

380. Preston, D. A. 1946. Legume diseases previously unreported from Oklahoma. Plant Dis. Reptr. 30: 45.
Records Phomopsis sojae, Phyllosticta glycineum, and Sclerotium rolfsii on soybean.

381. Reinking, O. A. 1918. Philippine economic-plant diseases. Philipp. Jour. Sci. 13: 165-274.
Including the following diseases on soybean: black mildew caused by Trotteria venturioides, Rhizoctonia blight, Peronospora trifoliorum, and Uromyces sojae.

382. _____. 1919. Host index of diseases of economic plants in the Philippines. Philipp. Agr. 8: 38-54.
Lists Peronospora trifoliorum, Rhizoctonia sp., Sclerotium sp., Trotteria venturioides, and Uromyces sojae, on soybeans.

383. _____. 1919. Diseases of economic plants in southern China. Philipp. Agr. 8: 105-135.
Including Uromyces sojae and Peronospora trifoliorum on soybean.

384. _____. 1919. Plant diseases in the Philippines. Phytopath. 9: 114-140.
Records Trotteria venturioides, Rhizoctonia sp., and Uromyces sojae on soybean.

385. Rhoads, A. S. 1944. Summary of observations on plant diseases in Florida during the emergency plant disease prevention project surveys, July 25 to December 31, 1943. Plant Dis. Reptr. Suppl. 148: 262-276.
Frog-eye disease and bacterial pustule caused defoliation, and anthracnose commonly attacked the pods. Failure to secure seed production constituted the greatest drawback to soybean culture.

386. Saccardo, P. A. 1917. Notae mycologicae. Ser. XXIII. Fungi Philippinenses a cl. Prof. C. F. Baker collecti et communicati. Atti del'Accad. Veneto-Trentino-Istriana 10: 57-94.
Including a technical description of Trotteria venturioides n. sp.

387. Samson, R. W. 1942. Tobacco ring spot on edible soybeans in Indiana in 1941. Plant Dis. Reptr. 26: 382.
A brief note on the occurrence of the disease.

388. Sasaki, S. 1929. (Mummy disease or black spot of soybean.) Ann. Agr. Exp. Sta. Gov.-Gen. Chosen 4: 1-28. (In Japanese).
The disease is due to a Phomopsis, the imperfect stage of Diaporthe sojae. Symptoms and morphology of the fungus are fully described.

389. Săvulescu, T., A. Aronescu, C. Sandu-Ville, and A. V. Alexandri. 1937. Phytosanitary conditions in Rumania during the year 1935-36. Inst. Cerc. Agron. României Publ. 38: 1-70.
A virus disease was found on soybean characterized by curling of the leaves, and by brown or yellow mosaic.

390. _____, C. Sandu-Ville, A. Aronescu, and A. V. Alexandri. 1936. Phytosanitary conditions in Rumania during the year 1934-35. Inst. Cerc. Agron. României Publ. 25: 1-97.
Soybean suffered important losses from three forms of virus disease: leaf curl, brown mosaic, and yellow mosaic. Inoculation was successful only with yellow mosaic and its properties were studied.

391. _____, _____, T. Rayss, and V. Alexandri. 1935. Phytosanitary conditions in Rumania during the year 1933-34. Inst. Cerc. Agron. României Publ. 24: 1-59.
Occurrence of mosaic on soybean.

392. Sawada, K. 1919. Descriptive catalogue of the Formosan fungi. Part 1. Agr. Exp. Sta. Gov't Formosa Spec. Bull. 19: 1-695. (In Japanese).
Including descriptions of the following fungi on soybean: Hypochnus centrifugus, H. sasakii, Sclerotinia libertiana.

393. _____. 1922. Descriptive catalogue of the Formosan fungi. Part 2. Dept. Agr. Gov't Res. Inst. Formosa Rep. 2: 1-173. (In Japanese).
Including a description of Peronospora manshurica on soybean.

394. _____. 1928. Descriptive catalogue of the Formosan fungi. Part 4. Dept. Agr. Gov't Res. Inst. Formosa Rept. 33: 1-123. (In Japanese).
Including descriptions of Colletotrichum glycines and Phakopsora sojae n. comb. on soybean.

395. Schropp, W. 1938. Beiträge zur Kenntnis der Kalimangelerscheinungen bei einigen Öl- und Gespinstpflanzen. Ernähr. Pfl. 34: 165-170, 181-186.
Soybeans reacted to the absence of potash by brown spotting of the foliage and inward curling of the margins, the young leaves in addition displaying an abnormally dark discoloration.

396. Schwarz, M. B. 1927. (Preliminary results of a crop rotation test extending over several years on rice soil in connection with investigations on slime disease (Bacterium solanacearum) in Arachis hypogaea.) Korte Meded. Inst. voor plantenziekten 3, 11 pp. (In Dutch).
Soybeans included in the rotation were attacked by the disease.

397. Sharp, C. G. 1927. Correlation of virulence and acid agglutination of a smooth and a rough strain of Bacterium phaseoli sojense. Phytopath. 17: 49.
Roughness in culture is correlated with lesser virulence and greater

407. Sommer, Anna L., J. I. Wear, and Aaron Baxter, 1940. The response to magnesium of six different crops on sixteen Alabama soils. Proc. Soil Sci. Soc. Am. 5: 205-216
There is a close relationship between manganese chlorosis and iron chlorosis. With a low iron:manganese ratio, iron deficiency (manganese toxicity) is developed. With a high ratio, iron toxicity (manganese deficiency) is developed.

408. Sprague, R. 1942. Soybean diseases in western North Dakota. Plant Dis. Reptr. 26: 382.
Bacterial blight was common. Fusarium scirpi var. acuminatum and Pythium debaryanum were frequently isolated from soybean seeds.

409. Steckel, J. E. 1946. Manganese fertilization of soybeans in Indiana. Proc. Soil Sci. Soc. Amer. 11: 345-348.
Manganese deficiency was found in soybeans that grew on soils with a high organic-matter content and a relatively high water-table.

410. _____. 1948. Manganese-deficient soybeans in Indiana. Soybean Dig. 8 (8): 14-15.
A yellow, sickly appearance of soybeans was noticed in fields for several years. Rapid recovery followed spraying with manganese sulfate. Soil application of manganese significantly increased the yield and spray application was equally effective.

411. Stevenson, J. A. 1926. Foreign plant diseases. 198 pp. U. S. Dept. Agr. Office of the Secretary.
Lists 19 diseases attacking Soja spp., which are new to or not widely distributed in the United States.

412. Steyaert, R. L. 1934. Observations sur la stigmatomycose des capsules du cotonnier au Congo Belge. Bull. Agr. Congo Belge 25: 473-493.
Soybean is recorded as one of the alternate hosts of Nematospora coryli and N. gossypii.

413. _____. 1948. Contribution a l'étude des parasites des végétaux du Congo Belge. Bull, Soc. Bot. Belg. 80: 11-58.
Includes a record of Nematospora phaseoli on soybean.

414. Stoddard, D. L. 1945. Seed treatment. In Federal Exp. Sta. Puerto Rico Rep. 1944. pp. 28-29.
Seed treatment of soybean with Arasan, Semesan, and Spergon gave protection against seed rots and pre-emergence damping-off.

415. Stone, G. M., and J. L. Seal. 1944. Plant diseases observed in Alabama in 1943. Plant Dis. Reptr. Suppl. 148: 276-280.
Mosaic and southern blight were prevalent on soybeans. Bacterial leaf spot, downy mildew, pod and stem blight, and frog-eye leaf spot also caused moderate damages.

416. Stubbs, M. W. 1936. Viroses of the garden pea, Pisum sativum. Phytopath. 26: 108-109.
An abstract of the following entry.

417. _____. 1937. Viroses of garden pea, Pisum sativum. Phytopath. 27: 242-266.
Pea virus 1 can infect soybean.

418. Supreme Commander of the Allied Powers. 1949. Japanese natural resources; a comprehensive survey. 559 pp. Tokyo, Japan.
Anthracnose of soybean is listed under "Major food crop diseases".

419. Suzuki, K. 1921. (Studies on the cause of purple seed of soybeans.) Rep. Chosen Agr. Ass'n. 16: 24-28.

The cause of the disease is attributed to the mechanical pressure exerted by the pod in consequence of unequal rate of growth between the pod and seed. Climatic conditions are considered as a chief factor affecting the disease.

420. Sydow, H. & P., and E. J. Butler. 1906. Fungi Indiae orientalis. Pars I. Ann. Myc. 4: 424-445.
Records the occurrence of the teleuto-stage of Uredo sojae, to which the name Uromyces sojae is given.

421. _____, and _____. 1916. Fungi Indiae orientalis. Pars V. Ann. Myc. 14: 177-220.
Contains a technical description of Septoria sojae n. sp.

422. Tai, F. L. 1936. Notes on Chinese fungi. VII. Bull. Chinese Bot. Soc. 2: 45-66.
Contains a description of Cercospora daizu.

423. _____, and C. T. Wei. 1933. Notes on Chinese fungi. III. Sinensia 4: 83-128.
Contains a description of Peronospora manshurica.

424. Takasugi, H. 1936. Division of Plant Pathology and Entomology. Contr. Agr. Exp. Sta. Manchurian R. R. 1933, pp. 583-738. (In Japanese)
Including notes on the occurrence and prevalence of the following organisms on soybean: Bacterium sojae var. japonicum, Peronospora manshurica, Cercospora kikuchii, Septoria glycines, Pleosphaerulina sojaecola, Gibberella sp., and Cercospora sojina.

425. Takimoto, S. 1916. (Two diseases of soybean.) Jour. Pl. Prot. (Tokyo) 3: 368-369. (In Japanese)
Records downy mildew and another disease due to mite injury.

426. _____. 1921. (Bacterial spotting disease of soybean.) Jour. Pl. Prot. (Tokyo) 8: 237-241. (In Japanese)
A preliminary note of the following entry. The causal organism is not named.

427. _____. 1927. (Bacterial spotting disease of soybean.) Jour. Pl. Prot. (Tokyo) 14: 559-566. (In Japanese)
The disease occurred in Japan and Korea. Its causal organism is named Bacterium sojae var. japonicum n. var., and morphology and cultural characters are described in detail.

428. _____. 1931. Leaf-scorch and leaf spotting of soybean. Jour. Pl. Prot. (Tokyo) 18: 175-179. (In Japanese)
The disease is caused by Bacterium phaseoli var. sojense, and Pseudomonas glycines is considered a synonym.

429. Tanaka, T. 1921. (On soybean nematode; preliminary identification). Jour. Pl. Prot. (Tokyo) 8: 551-553 (In Japanese)
Describes the morhpology of Heterodera schachtii, with suggestions on its control.

430. Taylor, C. F. 1944. Emergency plant disease survey in Virginia, 1943. Plant Dis. Reptr. Suppl. 148: 233-238.
Occurrence of Alternaria sp., Cercospora sojina, Phyllosticta glycineum and Xanthomonas phaseoli var. sojense on soybean.

431. _____. 1944. Emergency plant disease survey in West Virginia in 1943. Plant Dis. Reptr. Suppl. 148: 239-294.
Occurrence of Xanthomonas phaseoli var. sojense on soybean.

432. Tehon, L. R., and G. H. Boewe. 1939. Charcoal rot in Illinois. Plant Dis. Reptr. 23: 312-325.
The disease was found on soybean.

433. _____, and E. Y. Daniels. 1927. Notes on the parasitic fungi of Illinois III. Mycologia 19: 110-129.
Includes a technical description of Phyllosticta glycineum n. sp.

434. Tervet, I. W. 1943. Molds injurious to soybean seed. Minnesota Farm & Home Sci. 1: 13-14.
Severe retardation in seedling growth resulted from storage conditions favoring the development of Aspergillus spp., but seed treatment with maximum adhesive load of Arasan improved the vigor and stand of plants.

435. _____. 1943. Soybean diseases in Minnesota. Plant Dis. Reptr. 27: 135-138.
Among 50 varieties and selections of soybean grown in Minnesota in 1942, none was immune from bacterial blight. The following ones were immune from mosaic or mottle leaf: F.P.I. nos. 79610 and 92470; Minn. sel. nos. 118, 120 and 123 of Wisc. Manchu no. 3; Minn. sel. no. 107. of Minn. Manchu; and Holland no. 11.

436. _____. 1944. Diseases of soybeans in Minnesota. Plant Dis. Reptr. 28: 387.
Bacterial pustule and mosaic were common.

437. _____. 1944. Soybean diseases in Minnesota. Plant Dis. Reptr. 28: 835.
Bacterial pustule, bacterial blight, bud blight, mosaic and a root rot were found.

438. _____. 1944. Diseases in South Dakota in 1943. Plant Dis. Reptr. Suppl. 149: 308-311.
Records the occurrence of bacterial pustule on soybean.

439. _____. 1945. The influence of fungi on storage, on seed viability and seedling vigor of soybeans. Phytopath. 35: 3-15.
Species of Alternaria were the most frequent occupants of soybean seed samples in Minnesota, followed by Fusarium spp. The incidence of damage by micro-organisms increased in proportion to the extent of frost injury. Other fungi found in seeds of high moisture content included: Aspergillus glaucus, A. flavus, A. ochraceus, A. niger, A. fumigatus, Chaetomium sp., Cephalothecium roseum, Cunninghamella echinulata, Rhizopus nigricans, and Penicillium sp. Aspergillus flavus was the species predominantly concerned in the retardation of seedling growth. Arasan treatment improved the vigor of seedlings and increased the stand.

440. _____, and C. T. Tsiang. 1946. Pathogenicity of isolates of Rhizoctonia solani on soybean. Phytopath. 36: 411.
Isolates from various hosts differed considerably in virulence, but there was no evidence of race-host specificity.

441. Thomas, H. R., and W. J. Zaumeyer. 1950. Red node, a virus disease of bean. Phytopath 40: 28-29.
An abstract of the following entry.

442. _____, and _____. 1950. Red node, a virus disease of beans. Phytopath 40: 832-846.
The virus produced necrosis on soybean in inoculation experiments.

443. Thornberry, H. H., and H. W. Anderson. 1940. Pink-root disease of onion and tomatoes. Plant Dis. Reptr. 24: 383-384.
Soybean was found to be a host of Phoma terrestris.

444. Tidd, J. S. 1944. Soybean diseases in Indiana and Illinois. Plant Dis. Reptr. 28: 957-958.
Records the prevalence of the following diseases: downy mildew; bacterial pustule, bacterial blight, bud blight, stem and pod blight and charcoal rot.

445. Timnick, Margaret B., V. G. Lilly, and H. L. Barnett. 1948. The influence of light and other factors upon the sporulation of Diaporthe phaseoli from soybean. Amer. Jour. Bot. 35: 804.
On vegetable juice agar only pycnidia were formed. Few perithecia formed on vitamin-free casein hydrolysate medium unless cultures were irradiated with ultraviolet light. Exposure of cultures to continuous illumination from a daylight fluorescent tube for 60 days allowed the development of abundant perithecia, but almost completely inhibited the formation of asci and ascospores.

446. Tisdale, W. H. 1921. Two sclerotium diseases of rice. Jour. Agr. Res. 21: 649-657.
Slight morphological difference was noticed among isolates of Sclerotium rolfsii from soybean, wheat, and Arrhenatherum elatius.

447. Tokunaga, Y., and Y. Hashioka. 1948. (A preliminary report on crop diseases of Hainan Island). Taiwan Agr. Res. Inst. Agr. Bull. 2: 131-134. (In Chinese)
Soybean was attacked by Phakopsora sojae, Septoria glycines, Peronospora manshurica, and Corticium centrifugum.

448. Tomoyasu, R. 1924. (The causal fungus of purple seed of soybean). Jour. Pl. Prot. (Tokyo) 11: 310-315. (In Japanese)
The fungus was identified as Cercosporina sp.

449. Tu, C. 1932. Notes on diseases of economic plants in South China. Lingnan Sci. Jour. 11: 489-504.
Includes Uromyces sojae on soybean.

450. Tucker, C. M. 1924. Report of the Plant Pathologist. Porto Rico Agr. Exp. Sta. Rep. 1923, pp. 15-16.
Soybeans were attacked by a disease causing a shrivelling of the seed in the pods.

451. Uppal, B. N., M. K. Patel, and M. N. Kamat. 1938. Bacterial leaf-spot of soybean in Bombay. Jour. Univ. Bombay 6: 16-18.
The organism causing the disease is identical with Phytomonas phaseoli var. sojense.

452. Van der Goot, P., and H. R. A. Muller. 1932. Pests and diseases of the soybean crop in Java. Preliminary report. Landbouw Tijdschr. Landb. Nederl.-Indië 7: 683-704 (In Dutch, with English summary pp. 758-759).
Diseases on soybeans in Java are mainly of minor importance. The following diseases have been found: Slime disease caused by Bacterium solanacearum, foot-rot caused by Sclerotium rolfsii and anthracnose caused by Colletotrichum glycines. Under wet conditions foot-rot may cause some losses.

453. Van der Wolk, P. C. 1916. Study of a bacterial disease of soybean and the nature of the root nodules of Glycine soja and Arachis hypogaea. Cultura 28: 268-285, 300-319.
The disease first appears as an etiolated condition and may result in the death of the plant. It was considered to be caused by the activities of the bacterium, Rhizobium beijerinckii, associated with root nodules.

454. Van Hall, C. J. J. 1921. (Diseases and pests of cultivated plants in Dutch East Indies during 1921). Meded. Inst. voor Plantenziekten 53, 46 pp. (In Dutch)
Records Bacterium solanacearum on soybean.

455. Vassilieff, A. A. 1933. (Wilt of cultivated bast-yielding plants under Central Asian conditions). In (Diseases and pests of new cultivated textile plants). pp. 22-24. Inst. New Bast Material Vaskhnil, Moscow. (In Russian)
Soybean became infected by Verticillium dahliae when sown in pots which previously bore severely infected cotton plants. The external symptoms on soybean are similar to those on cotton.

456. Vestal, E. F. 1944. Diseases in stored grain and soybeans in Iowa. Plant Dis. Reptr. 28: 184-186.
Germination of soybean seed samples varied from 86.8 to 90 percent. Fungi present on the seed had not yet been determined.

457. Wahl, von. 1921. Schädlinge an der Sojabohne. Zeitschr. Pflanzenkr. 31: 194-196.
Erysiphe polygoni and Sclerotinia libertiana were observed on soybean in Augustenberg, Germany.

458. Walker, E. A. 1944. Fungi obtained from stubble of soybeans and other legumes in the New Jersey-Delaware-Maryland area. Plant Dis. Reptr. 28: 686-687.
Phomopsis sojae and Macrophomina phaseoli were isolated.

459. _____. 1944. Soybean diseases in Maryland, Delaware and New Jersey. Plant Dis. Reptr. 28: 888-890.
Bacterial blight, Cercospora daizu, downy mildew, Septoria glycines, and a leaf spot caused by Phyllosticta sp. and Alternaria sp. were observed.

460. _____. 1944. Diseases observed on soybeans in New Jersey, Delaware and Maryland. Plant Dis. Reptr. 28: 1006-1008.
Records the prevalence of the following diseases: Bacterial blight, Phyllosticta leaf spot, Phomopsis stem canker, leaf spots caused by Cercospora canescens and C. sojina, brown spot, bacterial pustule and downy mildew.

461. _____. 1946. Soybean leaf spots in Maryland. Plant Dis. Reptr. 30: 333.
Records the occurrence of Cercospora leaf spot and Phyllosticta sojaecola.

462. Wallace, G. B., and Maud M. Wallace. 1945. Tanganyika Territory fungus list: Recent records, VI. Dept. Agr. Tanganyika Myc. Circ. 15: 1-2.
Includes a root disease of soybean caused by Rhizoctonia bataticola.

463. _____ and _____. 1947. Second supplement to the revised list of plant diseases in Tanganyika Territory. E. Afr. Agr. Jour. 13: 61-67.
Lists Ascochyta phaseolorum on soybean.

464. _____ and _____. 1949. A list of plant diseases of economic importance in Tanganyika Territory. Commonw. Myc. Inst. Myc. Papers. 26: 1-26.
Lists Ascochyta phaseolorum, Cercospora sp., Macrophomina phaseoli and a bacterial leaf spot on soybean.

465. Waterson, J. M. 1947. The fungi of Bermuda. Dept. Agr. Bermuda Bull. 23:
Records on soybean Macrophomina phaseoli, Peronospora manshurica, Phyllosticta sojaecola and Pseudomonas glycinea.

466. Watkins, G. M. 1944. Plant diseases observed in Texas during 1943. Plant Dis. Reptr. Suppl. 149: 326-338.
Reporting Macrophomina phaseoli, Phymatotrichum omnivorum, Pseudomonas glycinea and Xanthomonas phaseoli var. sojense.

467. Wehmeyer, L. E. 1933. The genus Diaporthe Nitschke and its segregates. 349 pp. Univ. Michigan Press.
Diaporthe sojae is renamed D. phaseolorum var. sojae.

468. Wei, C. T. 1934. Rhizoctonia sheath blight of rice. Coll. Agr. Univ. Nanking Bull. (N.S.) 15.

Rhizoctonia solani isolated from rice can infect soybean in inoculation tests.

469. _____. 1950. Notes on Corynespora. Commonw. Myc. Inst. Myc. Papers 34: 1-9.

Reduces Cercospora vignicola Kawam. and Helminthosporium vignae Olive to synonyms of Corynespora cassiicola (Berk & Curt.) Wei.

470. Weimer, J. L. 1947. Disease survey of soybean nurseries in the South. Plant Dis. Reptr. Suppl. 168: 27-53.

In the southern United States the most widespread diseases in 1944-1946 were bacterial pustule and bacterial blight. Other diseases observed included: wildfire, Cercospora leaf spot, mosaic, downy mildew, pod and stem blight, charcoal rot, sclerotial blight, anthracnose and leaf spot caused by Alternaria sp. or possibly arsenic injury. Varietal reaction toward the bacterial diseases, wildfire, and Cercospora leaf spot were noted.

471. _____. 1950. Blackpatch of soybean and other forage legumes. Phytopath. 40: 782-784.

The disease is caused by a sterile fungus which produces lesions on soybean and a number of other legumes, resembling those of frog-eye.

472. Weiss, Freeman. 1946. Check list revision. Plant Dis. Reptr. 30: 130-137.

Lists all the diseases, 47 in total, hitherto known to occur on soybeans in the United States.

473. Weiss, M. G. 1949. Soybeans. Adv. Agron. 1: 77-157.

Includes a review of recent literature, chiefly North American, on the following diseases: stem canker, stem and pod blight, brown stem rot, anthracnose, bud blight, charcoal rot, sclerotial blight, Fusarium blight, stem rot, root knot, bacterial blight, bacterial pustule, wildfire, brown spot, frog-eye disease, mosaic, downy mildew.

474. Welch, A. W. 1946. A study of soybean diseases and their control. Iowa Agr. Exp. Sta. Report on Agricultural Research for the year ending June 30, 1946. Part 1, pp. 191-193.

Seed treatment failed to give any beneficial effect. The ascogenous stage of Diaporthe sojae was observed on old stems overwintered in the field. The best medium for its pycnidial formation was sterilized soybean seed coats. When mixed with soil and held at 10° F. the bacterial pustule organism produced infection after 69 weeks and the bacterial blight organism after 45 weeks.

475. _____. 1947. Natural and cultural occurrence of the ascogenous stage of Diaporthe phaseolorum var. sojae. Phytopath. 37: 23.

The occurrence of the ascogenous stage of the fungus in nature was reported. The asci and ascospores produced on the host were smaller than those produced in culture.

476. _____. 1947. A study of soybean diseases and their control. Iowa Agr. Exp. Sta. Report on Agricultural Research for the year ending June 30, 1947. Part 1, pp. 170-171.

Investigations of the pod and stem blight disease resulted in the differentiation of two types of diseases. The pod and stem blight is caused by Diaporthe phaseolorum var. sojae, a heterothallic species. The stem canker is caused by Diaporthe arctii, a homothallic and more virulent species. Pythium and Rhizoctonia caused serious damage in the greenhouse under controlled conditions. Other diseases and organisms

observed included 3 species of Glomerella, brown spot, bacterial blight, bacterial pustule, Septoria brown spot, mosaic, bud blight, downy mildew and Sclerotinia blight.

477. Welch, A. W., and J. C. Gilman. 1948. Hetero-and homothallic types of Diaporthe on soybeans. Phytopath. 38: 628-637.
Two forms of Diaporthe were found on soybeans, differing in pathogenicity and types of perithecial development. D. phaseolorum var. sojae is heterothallic with scattered single perithecia, produces typical Phomopsis conidia and causes wilting. D. phaseolorum var. batatatis is homothallic with caespitose perithecial clusters, lacks the conidial stage and causes stem canker mainly on mature plants.

478. Willis, L. G., and H. B. Mann. 1930. Manganese as a fertilizer. Experiments on South Atlantic Coastal Plain soils. Amer. Fertil. 72: 2125.
Soybean grown on soils of North Carolina, which were heavily limed to counteract their natural acidity, suffered from three distinct types of chlorosis, caused by magnesium, potash, and manganese deficiencies.

479. Withrow, A. P., and J. P. Biebel. 1944. Nicotine fumigation injury in Biloxi soybean. Phytopath. 34: 256-257.
A severe chlorosis was developed on soybean following nicotine fumigation. The symptoms were experimentally produced.

480. Wolf, F. A. 1920. Bacterial blight of soybean. Phytopath. 10: 119-132.
A bacterial blight found in North Carolina is considered to differ from the one due to Bacterium glycineum. The organism is described as Bacterium sojae n. sp. Infected seeds are the chief means of overwintering and dissemination. Infected leaves left in the field also harbor the pathogene.

481. _____. 1922. Additional hosts for Bacterium solanacearum. Phytopath. 12: 98-99.
Soybean was found to be a natural host.

482. _____. 1922. Studies on fermentation of rare sugars by plant pathogenic bacteria. Jour. Elisha Mitchell Sci. Soc. 38: 12-13.
Bacterium glycineum and B. sojae can be differentiated by their specialized fermentative action on rare sugars. The former attacks manitol and galactose, while the latter does not.

483. _____. 1923. Studies on the physiology of some plant pathogenic bacteria. VII. Pectic fermentation in culture media containing pectin. Phytopath. 13: 381-384.
Pectic fermentation was demonstrated in Bacterium sojae and other bacteria.

484. _____. 1924. Bacterial pustule of soybean. Jour. Agr. Res. 29: 57-68.
The pathogene gains entrance through the stomata and passes into the intercellular spaces. The pustules arise by hypertrophic changes of any of the parenchymatous tissue.

485. _____, and A. C. Foster. 1921. Thermal death points of some bacterial plant pathogenes in relation to reaction of the media. North Carolina Agr. Exp. Sta. Tech. Bull. 20: 21-24.
The concentration of hydrogen ions is an agent of great importance in cellular destruction at high temperatures in Bacterium glycineum, B. sojae, and four other bacteria.

486. _____, and S. G. Lehman. 1924. Report of Division of Plant Pathology. North Carolina Agr. Exp. Sta. Ann. Rept. 47: 83-85.
Soybean in North Carolina was subject to at least 12 diseases, of which about half were constantly recurring and of major importance.

Peronospora sojae is seed-borne and differs from P. trifoliorum in morphology and parasitism.

487. Wolf, F. A., and S. G. Lehman. 1926. Brown spot disease of soybean. Jour. Agr. Res. 33: 365-374.
The disease and the causal fungus, Septoria glycines, are described. Infection occurs through the stomata and the mycelium is intercellular in the tissue. Considerable difference was noticed in varietal susceptibility in soybean.

488. _____, and _____. 1926. Diseases of soybeans which occur in North Carolina and the Orient. Jour. Agr. Res. 33: 391-396.
A brief account on the following diseases known to occur in both North Carolina and Eastern Asia: Wilt caused by Fusarium tracheiphilum, downy mildew, Septoria brown spot, pod and stem blight, anthracnose, Cercospora leaf spot, and bacterial blight. All except wilt and leaf spot proved to be seed borne. Bacterial pustule and Sclerotium rolfsii occur in North Carolina, but not in Asia. Sclerotinia libertiana, Hypochnus centrifugus, Uromyces sojae, and Pleosphaeulina sojaecola occur in Asia but not in North Carolina.

489. _____, and I. V. Shunk. 1921. Tolerance to acids of certain bacterial pathogenes. Phytopath. 11: 244-250.
Acetic acid is more toxic than other acids employed at the same pH, and a greater pH concentration in agar than in bouillon is required to inhibit growth. Bacterium glycineum and B. sojae are included as the test material.

490. Woods, M. W., and S. B. Fenne. 1942. Two new records for the frog-eye leaf spot. Plant Dis. Reptr. 26: 382-383.
Records the occurrence of the disease in Maryland and Virginia.

491. Woodworth, C. M. 1924. Mottling of soybeans. Jour. Hered. 15: 349-354.
The extent and expression of mottling on different plants are possibly controlled by genetic factors.

492. _____, and F. C. Brown. 1920. Studies on varietal resistance and susceptibility to bacterial blight of the soybean. Phytopath. 10: 68.
Of 47 varieties grown in 1918 in Wisconsin, about one half were completely resistant and the other half ranged from complete susceptibility to partial resistance to the disease.

493. Yamamoto, W. 1925. (A new disease of soybean.) Jour. Pl. Prot. (Tokyo) 12: 97-99. (In Japanese)
A new leaf spot was found in Japan. The causal fungus was tentatively referred to as Mycosphaerella sojae.

494. Yokogi, K. 1927. (On the Hypochnus disease of soybeans and its comparison with that of rice plants). Jour. Pl. Prot. (Tokyo) 14: 146-158. (In Japanese)
Both Hypochnus sasakii and H. centrifugus are known to be pathogenic to soybeans in Japan. Morphological studies and cross inoculation tests on the two fungi from both rice and soybean revealed no significant difference, and accordingly it was considered that only one species was involved. Optimum temperature for the growth of the fungus was found to be 30° C., for sclerotial development 28° C.

495. Yokogi, K. 1927. (Studies on the Hypochnus disease of Sesamum indicum and the pathogenicity of its causal organism to rice plants and soybeans). Agr. & Hort. 2: 487-500. (In Japanese)
Hypochnus centrifugus isolated from sesamum is pathogenic to soybean.

496. Yoshii, H. 1927. (Crop diseases in 1926). Ann. Agr. Exp. Sta. Chosen 7: 21-34 (In Japanese).
Including Phomopsis sp., Cercosporina kikuchii, brown seed, Septoria glycines, and mosaic on soybean.

497. _____, and S. Sasaki. 1926. (Mummy disease of soybean) Jour. Pl. Prot. (Tokyo) 14: 524-525.
Phomopsis sp. caused mummification of soybean seeds in Korea.

498. Young, P. A. 1944. Epidemic of charcoal rot of corn and other crops in East Texas. Plant Dis. Reptr. 28: 898-899.
Soybean was attacked by Macrophomina phaseoli.

499. _____. 1949. Symptoms and resistance of crop plants to charcoal rot and ashy stem blight. Phytopath. 39: 27.
Soybean was rated as very susceptible to Macrophomina phaseoli.

500. Yu, T. F. 1939. A list of plant viroses observed in China. Phytopath. 29: 459-461.
Includes mosaic on soybean.

501. Zaumeyer, W. J. 1938. A streak disease of peas and its relation to several strains of alfalfa mosaic virus. Jour. Agr. Res. 56: 747-772.
Lucerne mosaic virus can infect soybean but pea streak virus cannot.

502. _____, and L. L. Harter. 1943. Two new virus diseases of beans. Jour. Agr. Res. 67: 305-328.
Soybean was susceptible to both bean mosaic virus 4 and 4A by artificial inoculation.

503. _____, and H. R. Thomas. 1950. Yellow stipple, a virus disease of bean. Phytopath. 40: 847-859.
The virus produced mottling or local lesions on all varieties of soybeans tested.

NON-PARASITIC DISEASES

Nutritional Disorders

1. Boron deficiency
   Young leaves chlorotic between veins; downward curling of tips and crinkling of leaves; die-back of tips; flowering prevented; roots stunted.
   Literature: 113, 313, 331.

2. Boron toxicity
   A narrow margin of the leaf edge dies, with little preliminary yellowing. Leaf edge becomes thin.
   Literature: 342.

3. Calcium deficiency
   Leaves curl, veins turn brown; roots turn brown then die.
   Literature: 115, 141, 247, 313.

4. Iron deficiency
   Young leaves chlorotic between veins, progressing downward on plant.
   Literature: 115, 141, 218, 313, 341, 342, 407.

5. Leaf wrinkle
   Tip of the young leaf first becomes brown and later the edge shows injury for a third of its length. The leaf continues growth, but the dead areas prevent the blade from expanding and the leaf has a puckered appearance.
   Literature: 204.

6. Magnesium deficiency
   Leaves covered with small brown spots more marked on older leaves; such leaves soon shed; roots lose turgor.
   Literature: 115, 141, 313, 342, 478.

7. Manganese deficiency
   The areas between veins become pale green and then pale yellow. In severe cases, brown areas appear on the leaves which drop off prematurely.
   Literature: 115, 342, 407, 409, 410, 478.

8. Manganese toxicity
   Mild chlorosis and much crinkling of leaves with marginal cupping.
   Literature: 115, 407.

9. Nitrogen deficiency
   Leaves show bronze colored patches when or before light green color becomes evident; stalk slender; roots show thickening, injury and decay.
   Literature: 115, 141, 218, 313, 342.

10. Phosphorus deficiency
    Leaves show brown spots after flowering; seeds from solution cultures few, not viable; roots brownish.
    Literature: 141, 247, 313.

11. Potassium deficiency
    Irregular yellow mottling first appears around the edge of the leaflets. The chlorotic areas soon merge to form a continuous yellow border around the leaflets, and this is followed by marginal firing which may spread to half or more of the area of the leaflets. The chlorotic area soon dies and usually falls out.
    Literature: 92, 115, 117, 124, 141, 218, 246, 247, 294, 313, 342, 368, 395, 478.

12. Sulfur deficiency
    Leaves yellow, followed by development of brown spots; stems thinner and less succulent.
    Literature: 122, 141, 313.

13. Zinc toxicity
    In older leaves, the stalks of the simple leaves and of the leaflets bend sharply down but are not wilted, and a dark-red to almost black color appears along the midrib and the main veins. The younger leaves become pale green and the terminal bud frequently dies.
    Literature: 115, 123.

Injuries

1. Acid soil
    Literature: 247.

2. Arsenic injury
    Brown, often coalescing, spots with concentric rings appear on leaves.
    Literature: 218, 470.

3. Cold injury
    Literature: 196, 247, 305.

4. Drought
    Literature: 247.

5. Hail damage
    Literature: 36, 77, 129, 225, 226, 247.

6. Lightning injury
    Literature: 218.

7. Nicotine fumigation injury
    A severe permanent chlorosis appears at the leaf margins and interveinally. In older leaves, the chlorosis is less severe and the injured tissue becomes brown and necrotic.
    Literature: 479.

8. Purple seed
    Due to mechanical pressure exerted by the pod.
    Literature: 419.

9. Sunburn; sun scald
    Literature: 96, 326, 368.

PARASITIC DISEASES

Virus diseases

1. (Alfalfa mosaic virus) [1]
    Literature: 57, 58.

2. Aster-yellows virus
    Possibly causing phyllody.
    Distribution: U. S. A.
    Literature: 110.

---

[1] Parentheses indicate results of artificial inoculation.

3. (Bean red node virus)
   Literature: 441, 442.

4. (Bean top necrosis virus)
   Literature: 261.

5. Bean yellow mosaic virus; Phaseolus virus 2
   Distribution: U. S. A.
   Literature: 23, 97, 154, 370.

6. (Bean yellow necrosis virus)
   Literature: 223.

7. (Bean yellow stipple virus)
   Literature: 503.

8. (Canavalia mosaic virus)
   Literature; 403.

9. Cowpea mosaic virus
   Distribution: Trinidad
   Literature: 107.

10. (Cucumber mosaic virus; Cucumber virus 1)
    Literature: 152.

11. (Guar top necrosis virus)
    Literature: 84, 98.

12. (Ladino clover yellow patch virus)
    Literature: 249, 250.

13. (Lucerne mosaic virus)
    Literature: 370, 591.

14. (Pea enation mosaic virus; pea virus 1)
    Literature: 371, 416, 417.

15. (Pea streak virus)
    Literature: 82, 153.

16. (Southern bean mosaic virus 1)
    Literature: 502.

17. (Southern bean mosaic virus 2)
    Literature: 502.

18. Soybean mosaic virus; Soja virus 1 -- Mosaic
    Leaves are dwarfed, with margins curled downward and the surface puckered with dark green areas between veins and sometimes with chlorotic spotting. Petioles and internodes are shortened. Pods are stunted, flattened, and curved. Seed setting is delayed and reduced greatly.
    The virus is inactivated at temperatures from 64° to 66° C and by aging in vitro for 4-5 days.
    Distribution: Australia, Canada, China, Czechoslovakia, Germany, Italy, Japan, Roumania, Uganda, U. S. A.
    Literature: 40, 49, 50, 51, 55, 69, 78, 96, 97, 102, 114, 117, 125, 134, 150, 151, 155, 160, 170, 171, 172, 183, 184, 200, 201, 214, 218, 221, 227, 231, 232, 241, 245, 256, 257, 258, 260, 284, 300, 317, 326, 329, 334, 335, 358, 359, 368, 369, 371, 372, 389, 390, 404, 415, 436, 437, 470, 472, 473, 476, 496, 500.

19. (Subterranean clover mosaic virus)
Literature: 7.

20. Tobacco leaf curl virus
Distribution: Nyasaland
Literature: 22.

21. Tobacco ringspot virus -- Bud blight
Infection occurs prior to the completion of terminal elongation of the stem apex, showing as a bronzed appearance of young leaves and ultimately necrosis and brittleness of the apical growing point. Occasionally necrotic streaks occur on the petioles and large leaf veins. Infection near the flowering period results in the withering and dropping of young pod clusters and dark brown blotches on the remaining poorly developed pods.
Distribution: Canada, U. S. A.
Literature: 10, 12, 13, 69, 70, 80, 96, 102, 117, 184, 189, 200, 201, 216, 243, 317, 320, 387, 404, 437, 444, 472, 473, 476.

22. (Tobacco streak virus)
Literature: 130.

23. Tomato big bud virus
Distribution: U. S. A.
Literature: 108, 109.

Bacterial diseases

1. (Corynebacterium flaccumfaciens (Hedges) Dowson)
Literature: 169.

2. Erwinia lathyri (Manns & Taub.) Holland
Syn.: Bacillus lathyri Manns & Taub.
Distribution: England, U. S. A.
Literature: 299, 367.

3. Pseudomonas glycinea Coerper -- Bacterial blight
Syn.: Bacterium glycineum Coerper; B. sojae Wolf; Phytomonas glycinea Burkh.; P. sojae Burkh.; Pseudomonas sojae Stapp
Lesions first appear on cotyledons as brown spots, the tissue collapses and the seedlings may be killed. The infection later spreads to the leaves as small, angular, yellow spots that soon enlarge and become light brown and then dark brown to almost black. Diseased tissues eventually become dry and may drop out. Similar spots also occur on stems and pods.
Motile by one to several polar flagella; 1.2-1.5 x 2.3-3.0 μ; gram-negative; nutrient agar colonies white, smooth, glistening, convex, margin entire, butyrous, with or without browning of the medium.
Distribution: Australia, Bermuda, Brazil, Bulgaria, Canada, Czechoslovakia, Denmark, Germany, South Africa, U. S. A., U. S. S. R.
Literature: 10, 12, 16, 20, 42, 49, 50, 54, 67, 68, 69, 70, 73, 80, 89, 91, 93, 96, 104, 117, 125, 131, 160, 162, 163, 164, 165, 200, 201, 215, 218, 227, 229, 230, 232, 233, 241, 243, 247, 248, 255, 256, 260, 272, 284, 292, 293, 317, 326, 327, 345, 348, 368, 373, 401, 402, 404, 408, 435, 437, 444, 459, 460, 465, 466, 470, 472, 473, 474, 476, 480, 482, 483, 485, 486, 488, 489, 492.

4. Pseudomonas glycinea Coerper var. japonicum (Takimoto) Burkh.
Syn.: Bacterium glycineum var. japonicum Elliott; B. sojae var. japonicum Takimoto; Phytomonas glycinea var. japonica Magrou
Lesions appear first as dark green, water-soaked spots on the under surface of leaves. These increase in size and gradually become brown to black, angular spots. Part of the infected leaves may die. Linear spots may occur on petioles and spots on pods are brown to blackish and sunken.
Motile by 1 to 4 polar flagella, 0.6-0.8 x 1.6-3.0 μ. Differs also slightly from the species in length of chains, action in milk, and color in media.

Distribution: China, Japan, Korea.
Literature: 160, 214, 323, 352, 424, 426, 427.

5. Pseudomonas phaseolicola (Burkh.) Dowson
Syn.: Pseudomonas medicaginis Sackett var. phaseolicola Burkh.
Distribution, Australia, Sweden, U. S. A.
Literature: 2, 24, 116.

6. Pseudomonas solanacearum E. F. Sm.
A vascular disease; causes dwarfing, sudden wilting, shriveling of foliage, and a brown stain of vascular bundles.
Distribution: Indonesia, U. S. A.
Literature: 144, 396, 406, 452, 454, 481.

7. (Pseudomonas syringae van Hall)
Syn.: Phytomonas vignae (Gardner & Kendr.) Bergey et al. var. leguminophila Burkh.
Literature: 74.

8. Pseudomonas tabaci (Wolf & Foster) Stevens -- Wildfire
Lesions on leaves vary in size and are surrounded by a wide yellow halo under moist environments. The restricted type of spot with indistinct halo is usually dark brown in contrast to the light brown and more extensive type of lesions. When beating rains are frequent, lesions spread rapidly and coalesce to involve the entire leaflet.
Distribution: U. S. A.
Literature: 10, 42, 51, 53, 54, 59, 87, 102, 117, 257, 258, 259, 320, 404, 470, 472, 473.

9. (Pseudomonas viridiflava (Burkh.) Clara)
Syn.: Phytomonas viridiflava Burkh.
Literature: 74.

10. Rhizobium beijerinckii Hiltner & Störmer
Distribution: England
Literature: 373, 453.

11. Xanthomonas phaseoli (E. F. Sm.) Dowson
Syn.: Pseudomonas phaseoli E. F. Sm.
Distribution: Germany
Literature: 247.

12. Xanthomonas phaseoli (E. F. Sm.) Dowson var. sojense (Hedges) Starr & Burkh. -- Bacterial pustule
Syn.: Bacterium phaseoli var. sojense Hedges; B. glycines Elliott; Phytomonas phaseoli var. sojense Burkh.; P. glycines Magrou; Pseudomonas glycines Nakano
Lesions on leaves appear first as small, inconspicuous pale green or reddish brown spots slightly elevated in the center. The spots later change to angular and reddish brown with marginal yellowing under some conditions. The spots may remain small or fuse to form large irregular mottled brown areas involving a large part of the leaf. Parts of spots may fall off leaving the leaf ragged. Small, elevated, reddish brown spots also occur on pods of susceptible varieties.
Motile by 1-2 polar flagella; 0.5-0.9 x 1.4-2.3 μ; gram-negative; beef agar colonies small, circular or nearly circular, smooth, pale yellow, margin entire, becoming deeper yellow with age and then often pale ringed, non-viscid to slightly viscid and with internal convolutions.
Distribution: Australia, China, Germany, India, Japan, Lithuania, U. S. A., U. S. S. R.
Literature: 4, 10, 12, 17, 25, 28, 42, 49, 51, 52, 68, 69, 70, 72, 80, 89, 102, 116, 117, 162, 166, 167, 168, 214, 218, 227, 233, 241, 243, 256, 257, 258, 259, 260, 265, 266, 269, 271, 272, 273, 278, 282, 289, 301, 302, 303, 306, 311, 326, 333, 334, 335, 345, 347, 351, 357, 358, 359, 368, 369, 372, 385, 397, 398, 404, 415, 428, 430, 431, 436, 437, 438, 444, 451, 460, 466, 470, 472, 473, 476, 484, 486, 488.

13. (Xanthomonas vesicatoria (Doidge) Dowson)
Literature: 116.

Fungus Diseases

1. Aecidium glycines P. Henn.
   Distribution: Africa.
   Literature: 114 (Originally described on Glycine javanica L.)

2. Alternaria atrans Gibson
   A secondary parasite following injury by aphids or sunburn.
   Distribution: China, U. S. A.
   Literature: 139, 291, 472.

3. Alternaria sp. -- Alternaria leaf spot
   Leaf spots relatively large and brown, with concentric rings.
   Distribution: Australia, China, U. S. A.
   Literature: 69, 80, 218, 256, 287, 314, 347, 368, 430, 459, 470.

4. Alternaria sp.
   Seed infestant
   Distribution: U. S. A.
   Literature: 218, 439.

5. Ascochyta phaseolorum Sacc.
   Distribution: Tanganyika.
   Literature: 463, 464.

6. Ascochyta pinodella L. K. Jones
   Distribution: Germany.
   Literature: 350.

7. Ascochyta pisi Lib.
   Distribution: France, Italy.
   Literature: 86, 111.

8. Ascochyta sojae Miura
   Spots on leaves, elliptical or irregular, 0.1-1 cm. diam., brown, becoming grayish and limited by elevated dark lines.
   Pycnidia amphigenous, dark brown, 90-120 μ diam.; spores fusiform, long ellipsoid, 12-18 x 4-4.5 μ.
   Distribution: China, Japan.
   Literature: 160, 214, 287.

9. Ascochyta sojaecola Abramoff
   Spots on leaves, stems, and pods, gray with brown margin, 0.5-2 cm. diam.
   Pycnidia slightly sunken, spheroidal, 90-220 μ diam.; spores hyaline, cylindrical or slightly ellipsoidal, 8-11 x 3-5 μ.
   Distribution: Belgian Congo, Japan, U. S. S. R.
   Literature: 1, 178, 210, 400.

10. Ascochyta sp.
    Spots on pods. Pycnidia 50-100 μ diam., ostiolate; spores 6-10 x 3-4 μ.
    Distribution: Japan, U. S. S. R.
    Literature: 160, 293.

11. Ascochyta sp.
    Spots on leaves, with concentric rings. Pycnidia 80-150 μ diam., ostiolate; spores 1-2 celled, 3-5 x 1.5-2 μ.
    Distribution: Japan.
    Literature: 158, 160.

12. Ascochyta sp.
    Distribution: Netherlands.
    Literature: 29.

13. Aspergillus spp.
Seed infestants.
Distribution: U. S. A.
Literature: 321, 434, 439, 472.

14. Botrytis cinerea Pers.
Distribution: Germany, U. S. A.
Literature: 89, 360, 361, 472.

15. Botrytis sp.
Distribution: U. S. S. R.
Literature: 292.

16. Cephalosporium gregatum Allington & Chamberlain
Causing browning of the pith and xylem of the stem, starting at or below the soil level and progressing slowly upward. Occasionally early blighting of the lower leaves occurs and is followed by a rapid interveinal chlorosis of upper leaves and subsequent necrosis. In advanced stages, the outside of the stem appears brown and the weakened stems lodge badly.
Conidia ovoid to elliptical, hyaline, continuous, 3.4-7.6 x 1.7-3.4 μ in pure culture, 6.8-9.4 x 3.4-4.3 μ on host; conidiophores hyaline, straight or clavate, chiefly 4-15 μ long.
Distribution: Canada, U. S. A.
Literature: 12, 14, 15, 41, 80, 81, 96, 100, 102, 103, 117, 125, 184, 185, 244, 379, 472, 473.

17. Cercospora canescens Ell. & G. Martin
Distribution: U. S. A.
Literature: 51, 460, 472.

18. Cercospora cruenta Sacc.
Distribution: U. S. A.
Literature: 51, 114, 472.

19. (Cercospora dolichi Ell. & Ev.)
Literature: 315.

20. Cercospora sojina Hara -- Frog-eye disease; Cercospora leaf spot
Syn: Cercospora daizu Miura
Leaf spots amphigenous, irregular to circular, zonate, grayish brown, with purplish margins; stem lesions elongate, reddish when young, becoming brown, smoky gray or almost black with age; pod lesions smaller and less zonate than those on leaves, usually on late-maturing varieties.
Conidia cylindrical, hyaline, 3-7 septate, 50-75 x 5-7 μ; conidiophores pale sooty colored, 0-3 septate, 60-150 x 4-6 μ.
Distribution: Australia, Canada, China, Germany, Japan, U. S. A., U. S. S. R.
Literature: 1, 6, 21, 40, 42, 49, 50, 51, 54, 69, 95, 117, 126, 157, 160, 214, 218, 247, 256, 257, 258, 259, 260, 264, 268, 269, 278, 287, 323, 326, 335, 368, 369, 385, 415, 422, 424, 430, 459, 460, 461, 470, 472, 473, 486, 488, 490.

21. Cercospora stevensii Young
Distribution: Brazil.
Literature: 328.

22. Cercospora spp.
Distribution: India, Tanganyika, U. S. A.
Literature: 162, 218, 322, 464.

23. Cercosporina kikuchii Mats. & Tomoy. -- Purple speck; purple stain
Spots on leaves, stems, pods and seeds, dark anthracene violet, irregular.
Conidia filiform, hyaline, 0-22 septate, 70-165 x 4-5 μ; conidiophores fasciculate, sooty brown, 85-220 x 4-6 μ, multiseptate.
Distribution: Canada, China, Japan, Korea, U. S. A.
Literature: 114, 132, 133, 146, 160, 214, 217, 239, 275, 287, 291, 309, 310, 314, 323, 324, 326, 335, 336, 368, 369, 424, 448, 472, 496.

24. Chaetomium sp.
Seed infestant.
Distribution: U. S. A.
Literature: 439.

25. (Colletotrichum indicum Dastur)
Literature: 288.

26. Colletotrichum pisi Pat.
Distribution: U. S. A.
Literature: 140.

27. Colletotrichum sp.
Distribution: China.
Literature: 287.

28. Colletotrichum truncatum (Schw.) Andrus & W. D. Moore
Distribution: U. S. A.
Literature: 140, 197.

29. Coniothyrium sojae Bouriquet
Lesions on the stem base. Pycinidia flattened, 108-150 μ diam.; stylospores cylindrical, non-septate, rounded at the end, 3-5.5 x 2-2.5 μ.
Distribution: Madagascar.
Literature: 66.

30. Corticium centrifugum (Lév.) Bres.
Syn: Hypochnus centrifugus Tul.
Causing stem canker.
Distribution: China, Japan, Korea, U. S. S. R.
Literature: 1, 160, 323, 336, 392, 447, 488, 494, 495.

31. Corticium sasakii (Shirai) Matsumoto
Syn: Hypochnus sasakii Shirai.
Distribution: China, Japan.
Literature: 209, 392, 494.

32. Corynespora cassiicola (Berk, & Curt.) Wei
Syn.: Cercospora vignicola Kawam.; Helminthosporium vignae Olive, Bain & Lefebvre; H. vignicola Olive.
Spots on leaves, small, brownish, not zoned.
Distribution: China, Japan, U. S. A.
Literature: 198, 291, 320, 353, 354, 469, 472.

33. Cunninghamella echinulata Thaxt.
Seed infestant.
Distribution: U. S. A.
Literature: 439.

34. Curvularia trifolii (Kauffman) Boedijn
Seed infestant.
Distribution: Canada.
Literature: 148.

35. Diaporthe phaseolorum (Cke. & Ell.) Sacc. var. batatatis (Harter & Field) Wehm. -- Stem canker

Large, light to dark brown lesions circumscribe the stems, causing eventually the wilting and death of the plant.

Distribution: U. S. A.

Literature: 102, 472, 473, 476, 477.

36. Diaporthe phaseolorum (Cke. & Ell.) Sacc. var. sojae (Lehm.) Wehm. -- Pod and stem blight.

Syn.: Diaporthe sojae Lehm.; Phomopsis sojae Lehm.

Dark brown lesions with indefinite margins occur on stems and pods and less commonly on leaves. Infection usually starts at the junction of a branch or petiole and girdles the stem, causing premature death of the plant.

Perithecia with long, cylindrical beaks, borne singly on effused stroma, erumpent, 156-260 x 192-335 μ; asci sessile, elongate, 8-spored, 37-52 x 7.4-12.9 μ; ascospores hyaline, ellipsoidal, 1-septate, 10.4-18.5 x 3.7-5.5 μ. Pycnidia lenticular to subglobose, ostiolate, 82-225 x 82-375 μ; conidiophores simple, tapering; conidia oblong, often fusiform, straight, hyaline, 6.3-7.2 x 2.2-2.3 μ; stylospores seldom present, hyaline, slender, curved or hooked.

Distribution: Canada, China, Japan, Korea, U. S. A., U. S. S. R.

Literature: 10, 30, 31, 34, 35, 53, 54, 69, 70, 94, 95, 96, 117, 126, 133, 145, 150, 151, 160, 183, 184, 217, 218, 240, 241, 243, 257, 258, 259, 260, 262, 263, 287, 291, 295, 296, 317, 318, 326, 359, 368, 373, 381, 388, 400, 415, 444, 445, 458, 460, 467, 470, 472, 473, 474, 476, 477, 486, 488.

37. Diplodia sp.

Distribution: China.

Literature: 291.

38. Epicoccum neglectum Desm.

Distribution: U. S. S. R.

Literature: 400.

39. Erysiphe communis Fr.

Distribution: Europe.

Literature: 144.

40. Erysiphe polygoni DC. -- Powdery mildew

Dull gray spots occur on the upper surface of the leaves and pale pink to deep vinaceous coloring in the tissues.

Distribution: Germany, Peru, South Africa, U. S. A.

Literature: 27, 56, 117, 267, 457, 472.

41. (Fomes lignosus Klotzsch.)

Literature: 156.

42. Fusarium oxysporum Schl. f. tracheiphilum (E. F. Sm.) Snyder & Hansen -- Fusarium blight; wilt

Syn.: Fusarium tracheiphilum E. F. Sm.; F. bulbigenum Cke. & Mass. var. tracheiphilum (E. F. Sm.) Wr.

The root and the stem base show browning as well as the browning or blackening of the vascular bundles. The leaves become yellow and drop, and the pods are poorly developed. Lesions on the pods and seedling blight also occur.

Distribution: Canada, China, Germany, Japan, South Africa, U. S. A., U. S. S. R.

Literature: 1, 46, 47, 95, 96, 105, 106, 117, 125, 184, 217, 227, 241, 247, 256, 260, 287, 290, 326, 400, 472, 473, 486, 488.

43. Fusarium roseum Lk.

Syn.: Fusarium scirpi Lamb. & Fautr. var. acuminatum (Ell. & Ev.) Wr.

Distribution: Japan, U. S. A.

Literature: 158, 160, 408.

44. Fusarium solani (Mart.) Appel & Wr.
Syn.: Fusarium solani (Mart.) Appel & Wr. var. martii (Appel & Wr.) Wr.
Distribution: China.
Literature: 287.

45. Fusarium spp.
Distribution: Australia, Japan, U. S. A., U. S. S. R.
Literature: 1, 49, 218, 227, 292, 347, 349, 400, 439.

46. Gibberella sp.
Causing stem canker. Perithecia 170-260 x 150-220 µ; asci 48-84 x 8-12 µ; ascospores 24 x 4 µ.
Distribution: China, U. S. S. R.
Literature: 214, 227, 323, 424.

47. Gloeosporium sp.
Distribution: China, Japan, Korea.
Literature: 160, 175, 176, 291, 336.

48. Glomerella cingulata (Stonem.) Spauld. & Schrenk
Distribution: Europe.
Literature: 114.

49. Glomerella glycines Lehm. & Wolf. -- Anthracnose
Syn.: Colletotrichum glycines Hori.
Seedling blight occurs before or after emergence. Lesions appear as dark brown cankers on the cotyledons and hypocotyls. Lesions on stems, pods and seeds are indefinite brown areas. Diseased plants may die prematurely and pods fail to develop properly.
Perithecia membranaceous, rostrate, 200-340 µ diam.; asci oblong to bluntly clavate, 9.5-13.5 x 70-106 µ; ascospores hyaline, unicellular, 18-28 x 4-6 µ. Acervuli black, with numerous brown setae; conidia 20-22 x 4 µ.
Distribution: Canada, China, Germany, Indonesia, Japan, Korea, South Africa, U. S. A.
Literature: 27, 49, 51, 54, 94, 117, 140, 144, 160, 175, 176, 183, 208, 214, 218, 247, 257, 259, 281, 286, 287, 291, 302, 307, 317, 318, 326, 336, 359, 385, 394, 418, 452, 470, 472, 473, 476, 486, 488.

50. Helicobasidium purpureum (Tul.) Pat.
Causing root rot.
Distribution: Southern Rhodesia.
Literature: 205.

51. Helminthosporium sp.
Distribution: China.
Literature: 291.

52. (Helminthosporium victoriae Meehan & Murphy)
Literature: 316.

53. Isariopsis griseola Sacc.
Causing angular, brown spots on leaves.
Distribution: U. S. S. R.
Literature: 1.

54. Macrophoma mame Hara
Spots on pods, brown, depressed. Pycnidia globose or depressed, 132-170 µ diam., ostiolate; spores 15.4-25 x 6.6-8 µ.
Distribution: China, Japan.
Literature: 160, 214, 287, 291.

55. Macrophomina phaseoli (Maubl.) Ashby -- Charcoal rot
Syn.: Sclerotium bataticola Taub.
Causing rotting and hollowing of the stems and tap roots, rotting of the fruits, and wilting of the leaves.
Distribution: Bermuda, Canada, Ceylon, India, Southern Rhodesia, Tanganyika, U. S. A.
Literature: 49, 70, 186, 205, 217, 218, 243, 256, 257, 258, 259, 260, 285, 319, 359, 364, 368, 369, 432, 444, 458, 462, 464, 465, 466, 472, 473, 498, 499.

56. Microascus trigonosporus Emmons & Dodge
Distribution: U. S. A.
Literature: 118.

57. Microsphaera sp.
Distribution: U. S. A.
Literature: 274, 320.

58. Mycosphaerella phaseolicola (Desm.) Sacc.
Distribution: U. S. S. R.
Literature: 1.

59. Mycosphaerella phaseolorum Siem.
Distribution: U. S. S. R.
Literature: 400.

60. Mycosphaerella sojae Hori
Distribution: China, Japan, Korea,
Literature: 159, 160, 214, 287, 291, 336, 493. (Apparently no description had been published by Hori himself.)

61. Myrothecium roridum Tode
Causing leaf spot.
Distribution: U. S. A.
Literature: 54, 320, 472.

62. Nematospora coryli Pegl. -- Yeast spot
Distribution: Belgian Congo, South Africa, U. S. A.
Literature: 258, 260, 270, 320, 412, 472.

63. Nematospora gossypii Ashby & Nowell -- Yeast spot
Distribution: Belgian Congo, South Africa.
Literature: 325, 412.

64. Nematospora phaseoli Wingard -- Yeast spot
Distribution: Belgian Congo, U. S. A.
Literature: 270, 413.

65. Oidium balsamii Mont.
Distribution: Peru.
Literature: 56.

66. (Olpidium trifolii (Pass.) Schroet.)
Literature: 253.

67. (Olpidium viciae Kusano)
Literature: 253.

68. Ophionectria sojae Hara
Causing browning and shrivelling of the stem near the ground level, and eventually the death of the plant.
Perithecia globose or oval, 100-200 μ diam.; asci clavate, stipate, 68-88 x 11-13 μ; ascospores oblong or fusiform, 1-septate, hyaline, 30-52 x 3-4 μ.

Distribution: China, Japan.
Literature: 160, 287.

69. Peckia sp.
Causing stem blight.
Distribution: Japan.
Literature: 147, 160.

70. Penicillium sp.
Lesions on cotyledons and hypocotyls appear either as sunken, dark brown spots, or as soft, water-soaked dark areas.
Distribution: U. S. A.
Literature: 161, 320, 439, 472.

71. Peronospora manshurica (Naoum.) Syd. -- Downy mildew
Syn.: Peronospora trifoliorum D. By. var. manshurica Naoum.; P. sojae Lehm. & Wolf
Lesions appear first as indefinite chlorotic spots on the upper surface of the leaves. Mature lesions are well defined, grayish to dark brown, with chlorotic margins. In susceptible varieties, lesions spread rapidly over the leaf surface, causing yellowing and finally browning of the leaf. The interior of the pods and the seed coat are often incrusted by a grayish mass of mycelium and oospores. In case of systemic infection, stunting of all the aerial organs of the plant may occur.
Conidiophores hypophyllous, gray to pale violet, 240-500 x 5-9 μ; conidia oblong to nearly round, pale gray violet, 24-27 x 18-21 μ; oospores globose, smooth, hyaline to pale yellow, 20-28 μ diam.
Distribution: Bermuda, Canada, China, Denmark, Estonia, Germany, Japan, Korea, Sweden, U. S. A., U. S. S. R.
Literature: 1, 10, 26, 42, 49, 50, 51, 54, 62, 68, 69, 70, 93, 95, 96, 102, 114, 117, 121, 135, 136, 137, 150, 151, 160, 162, 183, 184, 200, 201, 212, 214, 217, 218, 219, 224, 227, 241, 247, 256, 257, 269, 279, 284, 287, 292, 293, 301, 302, 317, 323, 326, 334, 335, 336, 339, 368, 369, 372, 378, 393, 415, 423, 424, 425, 444, 447, 459, 460, 465, 470, 472, 473, 476, 486, 488.

72. Peronospora trifoliorum D. By.
Distribution: India, Philippines, South Africa.
Literature: 27, 75, 76, 381, 382, 383.

73. Phakopsora pachyrhizi Syd. -- Rust
Syn.: Phakopsora sojae Saw.; Uredo sojae P. Henn.
Distribution: China, Japan, Korea.
Literature: 160, 191, 192, 193, 194, 213, 214, 287, 394, 447.

74. Phoma terrestris Hansen
Distribution: U. S. A.
Literature: 251, 443.

75. Phomopsis sp.
Causing mummification of the seed.
Distribution: Japan.
Literature: 496, 497.

76. Phyllosticta glycines Thuem.
Distribution: India.
Literature: 76. (Orginally described on Glycine violacea Schneev. = Hardenbergia monophylla Benth.)

77. Phyllosticta glycineum Tehon & Daniels
Spots on leaves, subcircular, cinereous, purple-bordered.
Pycnidia globose to somewhat applanate, 90-170 μ diam., ostiolate; conidia

oblong to narrowly ellipsoid, hyaline to smoky, 4.5-7 x 2-2.5 μ.
Distribution: U. S. A.
Literature: 101, 103, 337, 380, 430, 433, 472.

78. Phyllosticta sp.
Distribution: China, U. S. A.
Literature: 334, 459.

79. Phymatotrichum omnivorum (Shear) Duggar
Distribution: U. S. A.
Literature: 218, 466, 472.

80. Pleosphaerulina sojaecola Miura -- Phyllosticta leaf spot
Syn.: Phyllosticta sojaecola Massal.
Spots on leaves, brown, angular, becoming grayish, zonate.
Perithecia globose, non-ostiolate, 100-110 μ diam.; asci broadly ellipsoid, rounded and thickened at the apex, 57-77 x 35-40 μ, sessile, 8-spored; ascospores oblong, hyaline, muriform, 21-32 x 9-11 μ. Pycnidia globose, 100-180 μ; conidia hyaline, oblong or clavate, 2-3 guttulate, 5-10 x 3-3.5 μ.
Distribution: Bermuda, Canada, China, Estonia, Germany, Italy, Japan, Korea, U. S. A., U. S. S. R.
Literature: 1, 51, 63, 95, 96, 114, 160, 214, 227, 283, 287, 291, 308, 320, 323, 332, 335, 336, 355, 400, 424, 460, 461, 465, 472, 488.

81. Pythium debaryanum Hesse -- Pythium root rot
Causing light brown, soft rot of the basal portion of the young stem and the root.
Distribution: U. S. A.
Literature: 117, 218, 280, 312, 318, 326, 375, 408, 472.

82. (Pythium graminicola Subr.)
Literature: 375.

83. Pythium spp.
Distribution: China, U. S. A.
Literature: 232, 241, 287, 314, 318, 377, 476.

84. Rhizoctonia solani Kühn -- Root rot
Causing rotting of the roots and the stems near or below the soil level, with white mycelial mats associated with lesioned tissues.
Distribution: China, Philippines, U. S. A.
Literature: 64, 65, 68, 80, 83, 102, 114, 218, 232, 236, 237, 287, 291, 312, 319, 330, 365, 366, 368, 440, 468, 472.

85. Rhizoctonia spp.
Distribution: India, Peru, Philippines, U. S. A.
Literature: 56, 112, 318, 340, 381, 382, 384, 476.

86. Rhizopus nigricans Ehr.
Seed infestant.
Distribution: U. S. A.
Literature: 439.

87. Sclerotinia sclerotiorum (Lib.) D. By. -- Stem rot
Syn.: Sclerotinia libertiana Fuckel
The stem is attacked and girdled near the soil level, resulting in the death of the plant above the girdle.
Distribution: Argentina, Canada, China, Germany, Japan, Sweden, U. S. A., U. S. S. R.
Literature: 1, 60, 114, 184, 214, 227, 284, 287, 293, 304, 323, 326, 360, 392, 457, 472, 473, 476, 488.

88. Sclerotium rolfsii Sacc. -- Sclerotial blight; southern blight

Causing a rotting at the base of the stem and the upper portion of the tap root, with large globose brown sclerotia developed on diseased tissues.

Distribution: Australia, China, Indonesia, Philippines, South Africa, Trinidad, U. S. A.

Literature: 10, 27, 32, 37, 48, 49, 52, 53, 54, 69, 71, 79, 104, 114, 117, 126, 144, 184, 218, 241, 256, 287, 347, 372, 380, 415, 446, 452, 470, 472, 473, 486, 488.

89. Septoria glycines Hemmi -- Brown spot; Septoria leaf spot

Spots on leaves, first brown or light reddish brown, slightly raised, angular, gradually turning to dark brown, and finally to blackish brown, amphigenous, usually 2-3 mm. diam. Lesions also develop on stems and pods as the plants approach maturity.

Pycnidia amphigenous, globose or conico-globose, 44-100 μ diam.; spores hyaline, filiform, occasionally guttulate, obscurely 0-3 septate, 21-52.5 x 1.4-2.1 μ.

Distribution: Canada, China, Germany, Japan, Korea, U. S. A., U. S. S. R.

Literature: 1, 5, 42, 80, 95, 96, 114, 117, 160, 162, 173, 174, 177, 183, 199, 200, 201, 214, 218, 247, 272, 301, 302, 323, 326, 336, 368, 424, 447, 459, 460, 472, 473, 476, 486, 487, 488, 496.

90. Septoria sojae Syd. & Butl.

Spots on leaves and stems, scattered or confluent, margin indistinct, brown, later turning gray, surrounded by yellowish zones, 2-5 mm. diam.

Pycnidia epiphyllous, innate, dark brown, 75-100 μ diam.; spores filiform, non-septate, 25-40 x 0.5-1 μ.

Distribution: India.

Literature: 76, 421.

91. Septoria sojina Thuem.

Spots on leaves and stems, irregular, yellowish, with deep purple margin.

Pycnidia epiphyllous, scattered; spores cylindrical or almost cuneiform, straight, 1-septate, 2-4 guttulate, hyaline, 12-18 x 4.5-5 μ.

Distribution: Austria, Germany, U. S. S. R.

Literature: 9, 117, 400.

92. Sphaceloma sp. -- Scab

Causing hyperplastic lesions on leaves, stems and pods.

Distribution: Japan.

Literature: 252.

93. (Streptomyces scabies (Thaxt.) Waksm. & Henrici)

Literature: 202, 203.

94. (Thielaviopsis basicola (Berk. & Br.) Ferr.)

Literature: 220, 296.

95. Trichothecium roseum Link

Syn.: Cephalothecium roseum Cda.

Distribution: U. S. A.

Literature: 439, 472.

96. Trotteria venturioides Sacc. -- Black leaf mildew

Appearing as small black specks on the upper surface of the leaves.

Pycnidia hypophyllous, globose, long-setose, black, minute, 80-120 μ diam., ostiolate; spores cylindrical-fusiform, curved, hyaline, 5-7 celled, 38-45 x 4.5 μ.

Distribution: Philippines.

Literature: 381, 382, 384, 386.

97. (Verticillium dahliae Kleb.)

Literature: 455.

98. Uromyces sojae (P. Henn.) Syd.
Distribution: Ceylon, China, India, Philippines.
Literature: 76, 114, 214, 323, 362, 381, 382, 383, 384, 420, 449, 488.
(Apparently all records on soybean should be referred to Phakopsora pachyrhizi.)

Nematode Diseases

1. Anguillulina pratensis (de Man) Goffart
Syn.: Tylenchus brachyurus Godfrey; Pratylenchus pratensis Godfrey.
Causing brown lesions on the roots.
Distribution: Hawaii.
Literature: 142, 143, 363.

2. Anguillulina dipsaci (Kühn) Grev. & v. Ben.
Distribution: U. S. A.
Literature: 90, 143.

3. Heterodera marioni (Cornu) Goodey -- Root knot
Syn.: Anguillula radicicola Greeff; Heterodera radicicola Mueller
Affected plants lack vigor and are stunted and pale in color. Death of the plant may result from severe attacks. Irregular galls are found over the entire root system.
Distribution: Australia, Germany, Hawaii, U. S. A.
Literature: 49, 99, 114, 125, 128, 143, 214, 298, 347, 363, 405, 473.

4. Heterodera schachtii Schmidt -- Chlorosis; yellow dwarf
Affected plants are stunted and the foliage has an unhealthy yellow color. The roots are poorly developed.
Distribution: China, Japan.
Literature: 43, 143, '160, 195, 206, 211, 228, 323, 429.

Diseases due to Parasitic Seed Plant

1. Cuscuta chinensis Lamb. -- Dodder
Distribution: China, Japan, Korea.
Literature: 160, 214, 323.

DISEASES DUE TO UNDETERMINED CAUSES

1. Blackpatch
Distribution: U. S. A.
Literature: 471.

2. Brown seed spot
Distribution: China, Japan, Korea.
Literature: 160, 214, 343, 344.

3. Crinkling
Distribution: U. S. A.
Literature: 89, 90.

4. Curly leaf
Distribution: Europe.
Literature: 247.

5. Dwarf
Distribution: Europe
Literature: 247.

6. Pod and stem spot
   Distribution: U. S. A.
   Literature: 358, 359.

7. Seed mottling
   Distribution: U. S. A.
   Literature: 119, 356, 491.

8. "Sick soil" disease
   Distribution: China.
   Literature: 323.

9. Stem break
   Distribution: U. S. S. R.
   Literature: 1.

10. White stem spot
    Distribution: U. S. A.
    Literature: 49.

11. Wilt
    Distribution: China.
    Literature: 335.

SEED TREATMENT

Literature: 18, 33, 35, 38, 44, 85, 103, 112, 126, 127, 179, 180, 181, 182, 183, 184, 187, 188, 190, 233, 234, 235, 242, 243, 277, 302, 312, 374, 376, 377, 399, 414.

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 205

FUNGICIDAL AND PHYTOTOXIC PROPERTIES OF 412 SYNTHETIC ORGANIC COMPOUNDS

Supplement 205 July 15, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

FUNGICIDAL AND PHYTOTOXIC PROPERTIES OF 412 SYNTHETIC ORGANIC COMPOUNDS[1]

By M. C. Goldsworthy, senior pathologist, Bureau of Plant Industry, Soils, and Agricultural Engineering, and S. I. Gertler, chemist, Bureau of Entomology and Plant Quarantine, U. S. Department of Agriculture, Beltsville, Maryland

Plant Disease Reporter
Supplement 205 July 15, 1951

A previous publication[2] discussed the fungicidal and phytotoxic properties of 506 synthetic organic chemicals. This paper gives the results of similar tests with 412 additional synthetic organic compounds.

The methods used and the objectives sought in this second study do not differ essentially from those described in the first paper. They consisted, in general, of preparing suspensions of the chemicals in water from which residues were produced on cover slips and on peach foliage. The residues dried on the cover slips were subjected to test as fungicides by seeding them with suspensions of the conidia of Monilinia fructicola (Wint.) Honey (peach brown rot pathogen) and Glomerella cingulata (Ston.) Spauld. & Schrenk (apple bitter rot pathogen) and determining the germination of the spores. In these tests as before, the cover slips bearing the residues were made in sufficient number so that some could be tested immediately in the laboratory and others could be tested after periods of exposure in an apple orchard environment. This exposure, accomplished by suspending in apple trees the cover slips clamped in special holders, permitted periodical determinations of the effect of weathering on the fungicidal property of each chemical. All the chemicals were subjected to at least one weathering test. If any were found to possess fungicidal properties, they were subjected to more than one weathering test.

Phytotoxic tests were conducted along with the fungicidal tests. After the residues had been prepared on the cover slips, the remaining suspension was sprayed on the leaves of peach trees. The sprayed foliage was examined during a period of several weeks to determine the accumulative effect of the orchard environment on phytotoxicity.

In the initial tests all the compounds were ground up in an equal quantity, by weight, of pyrophyllite to insure a more favorable particle size for suspension. When a compound proved to be promising, i.e., possessed fungicidal properties, it was formulated also with DDT, benzene hexachloride, or parathion to study its fungicidal and phytotoxic properties in combination with these commonly used organic insecticides.

The procedures described in the previous paper were modified slightly. Plastic cover slips were substituted for fragile glass cover slips. This change eliminated loss of test materials through wind breakage. A 50-50 mixture of Czapek's solution and beef bouillon was substituted for the dilute Czapek's solution used in the earlier work to promote spore germination.

Results

Screening tests showed that 388 (listed in Table 1) of the 412 compounds tested did not possess fungicidal properties.

---

[1]The Plant Disease Survey recognizes that the material presented in this Supplement differs from the usual content of the Plant Disease Reporter, and may not be of direct interest to many of its readers. It is issued as a special contribution because of its fundamental importance. The Survey does not assume responsibility for the subject matter. The expense of publication is borne by the Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering. P. R. M.

[2]Goldsworthy, M. C., and S. I. Gertler. Fungicidal and phytotoxic properties of 506 synthetic organic compounds. Plant Dis. Reptr. Suppl. 182: [89]-109. 1949.

The 24 chemicals found to possess fungicidal properties are listed in Table 2. Of these 24, nine were found to be too phytotoxic to warrant further consideration. Two of the remaining 15 (N-ethyl anisamide and 2, 3, 5-trichloro-6-phenyl-p-quinone) caused mild injury to peach foliage, but one of these (2, 3, 5-trichloro-6-phenyl-p-quinone) was sufficiently promising as a fungicide to warrant further tests. None of the remaining 13 compounds were phytotoxic but only four of these [N-ethyl-p-nitrobenzamide; carbonic acid, bis (p-nitrophenyl) ester; 2, 3, 6, 7-tetrachloro-5, 6, 7, 8-tetrahydronaphthoquinone; and 2, 2'-thiobis (4-chlorophenol)] appeared to be promising enough to be studied more intensively.

Table 3 shows the fungicidal and phytotoxic properties of 11 of the chemicals when formulated with pyrophyllite, DDT, benzene hexachloride, or parathion. In some cases duration of fungicidal effectiveness was less when a chemical was formulated with one of the insecticides than when it was formulated with pyrophyllite.

Of the 412 compounds tested, only the previously listed appeared to be of promise as fungicides. [These 5 are N-ethyl-p-nitrobenzamide; bis (p-nitrophenyl) ester of carbonic acid; 2, 3, 6, 7-tetrachloro-5, 6, 7, 8-tetrahydronaphthoquinone; 2, 2'-thiobis (4-chlorophenol); and 2, 3, 5-trichloro-6-phenyl-p-quinone.] Two of these, the bis (p-nitrophenyl) ester of carbonic acid and 2, 2'-thiobis(4-chlorophenol), have reached the orchard stage in their development. Both have been used on apple and peach varieties experimentally to control the diseases of these fruits. The phenol compound has been found to control apple scab, Brooks' spot, bitter rot, and sooty blotch, but it causes russeting of the fruit of the Golden Delicious, Stayman, York, Grimes Golden, and Delicious varieties of apples. On peaches it proved to be particularly promising in preliminary tests, since it appeared effective in controlling peach scab, brown rot, and, to some degree, bacterial spot without causing any injury to foliage or fruit. The carbonic acid derivative did not cause any leaf or fruit injury to apple or peach and appeared to be fungicidal under orchard conditions. So far we have not been able to procure sufficient quantities of the remaining three promising materials for orchard tests.

Table 1. Organic compounds having no fungicidal activity when applied as sprays formulated with an equal weight of pyrophyllite at the rate of one pound per 100 gallons. [a]

Chemical	Injury to peach leaves [b]
Acetaldehyde, 2-chloroethyl 2, 4, 5-trichlorophenyl acetal	0
Acetamide, alpha, alpha, alpha-trichloro-N-2-thiazolyl-	Very severe
Acetanilide, 3-chloro-alpha-(pentachlorophenoxy)-	0
Acetanilide, alpha, alpha, alpha, 4-tetrachloro-	0
Acetanilide, alpha, alpha, 4-trichloro-	0
Acetanilide, alpha, alpha, alpha-trichloro-p-sulfamyl-	0
Acetic acid, 2-(2, 4-dinitrophenyl) hydrazide	0
Acetic acid, (4, 6-dinitro-o-toloxy)-	0
Acetic acid, phenyl-, m-tolyl ester	0
Acetic acid, phenyl-, p-tolyl ester	0
Acetic acid, o-toloxy-	0
Acetic acid, alpha-(trichloromethyl)benzyl ester	0
Acetic acid, 1-trichloromethyl-2, 2'-methylenebis (4, 6-dichlorophenyl) ester	0
Acetic acid, 2, 4, 5-trichlorophenyl ester	0
Acetoacetic acid, alpha, alpha-bis(2-cyanoethyl)-, ethyl ester	0
Acetoacetic acid, alpha, alpha-bis(2-cyanoethyl)-, methyl ester	Slight
Acetone, 2, 4-dinitrophenylhydrazone	0
Acetophenone, 2, 4-dinitrophenylhydrazone	0
m-Acetotoluidide, alpha, alpha, alpha-trichloro-alpha^3, alpha^3, alpha^3-trifluoro-	Severe
Aniline, N-allyl-2, 4-dinitro-	0
Aniline, N-sec-butyl-2, 4-dinitro-	0
Aniline, 2, 5-diethoxy-	0
Aniline, 2, 5-dimethoxy-	0
Anisamide, N-allyl-	Slight
Anisamide, N-benzyl-	0
Anisamide, N-cyclohexyl-	0
Anisamide, N-isobutyl-	0
Anisamide, N-isopropyl-	0
Anisanilide	0
Anisanilide, 4'-bromo-	0
Anisanilide, 2'-chloro-	Slight

[a] The compounds in all tables are named in the inverted form as used in Chemical Abstracts index in order to group together chemically related compounds as far as possible.

[b] Slight = trace of intercostal or marginal necrotic areas.
Medium = few intercostal or marginal necrotic areas.
Moderate = appreciable spotting of leaves with necrotic areas.
Severe = heavy intercostal and marginal necrosis but no effect on leaf abscission.
Very severe = heavy intercostal and marginal necrosis with leaf abscission.

Table 1. (Continued)

Chemical	Injury to peach leaves[b]
Anisanilide, 3'-chloro-	0
Anisanilide, 4'-chloro-	0
Anisanilide, 2', 5'-dichloro-	0
Anisanilide, 2'-nitro-	Slight
Anisanilide, 4'-nitro-	0
o-Anisanisidide	0
p-Anisanisidide	0
Anisic acid, o-bromophenyl ester	0
Anisic acid, p-bromophenyl ester	0
Anisic acid, p-tert-butylphenyl ester	0
Anisic acid, o-chlorophenyl ester	0
Anisic acid, 2, 4-dichlorophenyl ester	0
Anisic acid, p-ethylphenyl ester	0
Anisic acid, o-nitrophenyl ester	0
Anisic acid, p-nitrophenyl ester	0
Anisic acid, pentachlorophenyl ester	0
Anisic acid, phenyl ester	Medium
Anisic acid, phenylhydrazide	Slight
Anisic acid, m-tolyl ester	0
Anisic acid, o-tolyl ester	0
Anisic acid, 2, 4, 6-tribromophenyl ester	0
Anisic acid, 2, 4, 6-trichlorophenyl ester	0
Anisole, 5-allyl-2-(2, 4-dinitrophenoxy)-	0
m-Anisotoluidide	0
o-Anisotoluidide	0
p-Anisotoluidide	0
Anthranilic acid, N-(p-chlorobenzoyl)-	0
Anthranilic acid, N-(2, 4-dichlorobenzoyl)-	0
Benzaldehyde, 4-(2, 4-dinitrophenoxy)-3-methoxy-	0
Benzaldehyde, 2, 4-dinitrophenylhydrazone	0
Benzamide, N-amyl-o-chloro-	0
Benzamide, N-sec-amyl-o-chloro-	0
Benzamide, N-amyl-p-chloro-	0
Benzamide, N-sec-amyl-p-chloro-	0
Benzamide, N-amyl-2, 4-dichloro-	Medium
Benzamide, N-sec-amyl-2, 4-dichloro-	Slight
Benzamide, N-amyl-3, 4-dichloro-	0
Benzamide, N-sec-amyl-3, 4-dichloro-	Moderate
Benzamide, N-benzoxy-	0
Benzamide, N-benzyl-o-chloro-	0
Benzamide, N-benzyl-p-chloro-	0
Benzamide, N-benzyl-2, 4-dichloro-	0
Benzamide, N-benzyl-3, 4-dichloro-	0
Benzamide, N-benzyl-m-nitro-	0
Benzamide, N-benzyl-p-nitro-	0
Benzamide, N-2-bicyclohexyl-p-chloro-	0
Benzamide, N-2-bicyclohexyl-2, 4-dichloro-	0
Benzamide, N-butyl-o-chloro-	0
Benzamide, N-sec-butyl-o-chloro-	0

Table 1. (Continued)

Chemical	Injury to peach leaves [b]
Benzamide, N-butyl-p-chloro-	0
Benzamide, N-sec-butyl-p-chloro-	0
Benzamide, N-butyl-2, 4, dichloro-	0
Benzamide, N-sec-butyl-2, 4, dichloro-	Slight
Benzamide, N-butyl-3, 4-dichloro-	0
Benzamide, N-sec-butyl-3, 4-dichloro-	0
Benzamide, o-chloro-	0
Benzamide, p-chloro-	0
Benzamide, o-chloro-N-cyclohexyl-	0
Benzamide, p-chloro-N-cyclohexyl-	0
Benzamide, p-chloro-N, N-diisobutyl-	0
Benzamide, p-chloro-N, N-diisopropyl-	0
Benzamide, o-chloro-N-ethyl-	0
Benzamide, p-chloro-N-ethyl-	0
Benzamide, p-chloro-N-heptyl-	0
Benzamide, p-chloro-N-sec-hexyl-	0
Benzamide, o-chloro-N-isobutyl-	0
Benzamide, p-chloro-N-isobutyl-	0
Benzamide, o-chloro-N-isopropyl-	0
Benzamide, p-chloro-N-isopropyl-	0
Benzamide, p-chloro-N-methyl-	0
Benzamide, o-chloro-N-propyl-	0
Benzamide, p-chloro-N-propyl-	0
Benzamide, N-cyclohexyl-p-nitro-	0
Benzamide, N, N-dibenzyl-o-chloro-	0
Benzamide, N, N-dibenzyl-p-chloro-	0
Benzamide, N, N-dibenzyl-3, 4-dichloro-	0
Benzamide, N, N-dibenzyl-m-nitro-	0
Benzamide, N, N-dibenzyl-p-nitro-	0
Benzamide, 2, 4-dichloro-	0
Benzamide, 3, 4-dichloro-	0
Benzamide, 2, 4-dichloro-N-cyclohexyl-	0
Benzamide, 3, 4-dichloro-N-cyclohexyl-	0
Benzamide, 3, 4-dichloro-N, N-diisobutyl-	0
Benzamide, 2, 4-dichloro-N, N-diisopropyl-	0
Benzamide, 3, 4-dichloro-N, N-diisopropyl-	0
Benzamide, 2, 4-dichloro-N, N-dimethyl-	0
Benzamide, 3, 4-dichloro-N, N-dimethyl-	0
Benzamide, 2, 4-dichloro-N-ethyl-	Medium
Benzamide, 3, 4-dichloro-N-ethyl-	0
Benzamide, 2, 4-dichloro-N-isobutyl-	0
Benzamide, 3, 4-dichloro-N-isobutyl-	0
Benzamide, 2, 4-dichloro-N-isopropyl-	0
Benzamide, 3, 4-dichloro-N-isopropyl-	0
Benzamide, 2, 4-dichloro-N-methyl-	0
Benzamide, 3, 4-dichloro-N-methyl-	0
Benzamide, 2, 4-dichloro-N-propyl-	0
Benzamide, 3, 4-dichloro-N-propyl-	0
Benzamide, N, N^1-ethylenebis/2, 4-dichloro-	0
Benzamide, N-(2-hydroxyethyl)-p-nitro-	Slight
Benzanilide, 4'-benzoyl-4-chloro-	0
Benzanilide, 4'-bromo-2-chloro-	0
Benzanilide, 4'-bromo-4-chloro-	0
Benzanilide, 2'-bromo-2, 4-dichloro-	0

Table 1. (Continued)

Chemical	Injury to peach leaves[b]
Benzanilide, 2'-bromo-3, 4-dichloro-	0
Benzanilide, 4'-bromo-2, 4-dichloro-	0
Benzanilide, 4'-bromo-3, 4-dichloro-	0
Benzanilide, 2-chloro-	0
Benzanilide, 4-chloro-	0
Benzanilide, 2-chloro-2'-nitro-	0
Benzanilide, 2-chloro-3'-nitro-	0
Benzanilide, 2-chloro-4-nitro-	0
Benzanilide, 2-chloro-4'-nitro-	0
Benzanilide, 3'-chloro-4-nitro-	0
Benzanilide, 4-chloro-2'-nitro-	0
Benzanilide, 4-chloro-3'-nitro-	0
Benzanilide, 4-chloro-4'-nitro-	0
Benzanilide, 4'-chloro-4-nitro-	0
Benzanilide, 4-chloro-4'-phenylazo-	0
Benzanilide, 2, 2'-dichloro-	0
Benzanilide, 2, 3'-dichloro-	0
Benzanilide, 2, 4-dichloro-	0
Benzanilide, 2', 4-dichloro-	0
Benzanilide, 2, 4'-dichloro-	0
Benzanilide, 3', 4-dichloro-	0
Benzanilide, 4, 4'-dichloro-	0
Benzanilide, 2, 4-dichloro-2'-nitro-	0
Benzanilide, 2, 4-dichloro-3'-nitro-	0
Benzanilide, 2, 4-dichloro-4'-nitro-	0
Benzanilide, 2', -5'-dichloro-4-nitro-	0
Benzanilide, 3, 4-dichloro-2'-nitro-	0
Benzanilide, 3, 4-dichloro-3'-nitro-	0
Benzanilide, 3, 4-dichloro-4'-nitro-	0
Benzanilide, 2, 4-dichloro-2'-phenyl-	0
Benzanilide, 2, 4-dichloro-4'-phenylazo-	0
Benzanilide, o'-hydroxy-	0
Benzanilide, 4-nitro-	0
Benzanilide, 2, 2', 4, 5'-tetrachloro-	0
Benzanilide, 2', 3, 4, 5-tetrachloro-	0
Benzanilide, 2, 2', 4-trichloro-	0
Benzanilide, 2, 2', 5'-trichloro-	0
Benzanilide, 2, 3, 4-trichloro-	0
Benzanilide, 2, 3', 4-trichloro-	0
Benzanilide, 2, 4, 4'-trichloro-	0
Benzanilide, 2', 4, 5'-trichloro-	0
Benzanilide, 3, 3', 4-trichloro-	0
Benzanilide, 3, 4, 4'-trichloro-	0
o-Benzanisidide, 2-chloro-	0
o-Benzanisidide, 4-chloro-	0
p-Benzanisidide, 2-chloro-	0
p-Benzanisidide, 4-chloro-	0
o-Benzanisidide, 2, 4-dichloro-	0
p-Benzanisidide, 2, 4-dichloro-	0
p-Benzanisidide, 3, 4-dichloro-	0
Benzene, 1, 3-bis(2, 4-dinitrophenoxy)-	0
Benzene, 1, 4-bis(2, 4-dinitrophenoxy)-	0

Table 1. (Continued)

Chemical	Injury to peach leaves[b]
Benzenesulfonamide, 3, 4-dichloro-N-methyl-	0
Benzoic acid, o-benzamidophenyl ester	0
Benzoic acid, benzylidenehydrazide	0
Benzoic acid, p-chloro-, pentachlorophenyl ester	0
Benzoic acid, o-chloro-, phenylhydrazide	0
Benzoic acid, cinnamylidenehydrazide	0
Benzoic acid, 2, 4-dichloro-, p-bromophenyl ester	0
Benzoic acid, 2, 4-dichloro-, o-chlorophenyl ester	0
Benzoic acid, 3, 4-dichloro-, o-chlorophenyl ester	0
Benzoic acid, 2, 4-dichloro-, o-nitrophenyl ester	0
Benzoic acid, 3, 4-dichloro-, o-nitrophenyl ester	0
Benzoic acid, 2, 4-dichloro-, pentachlorophenyl ester	0
Benzoic acid, 3, 4-dichloro-, pentachlorophenyl ester	0
Benzoic acid, 2, 4-dichloro-, phenylhydrazide	0
Benzoic acid, 3, 4-dichloro-, phenylhydrazide	0
Benzoic acid, o-(2, 4-dinitrophenoxy)-	0
Benzoic acid, alpha-methylbenzylidenehydrazide	0
Benzoic acid, p-nitro-, benzylidenehydrazide	0
Benzoic acid, p-nitro-, butylidenehydrazide	0
Benzoic acid, p-nitro-, p-tert-butyliphenyl ester	0
Benzoic acid, p-nitro-, o-chlorobenzylidenehydrazide	0
Benzoic acid, p-nitro-, cinnamylidenehydrazide	0
Benzoic acid, p-nitro-, cyclohexylidenehydrazide	0
Benzoic acid, p-nitro-, cyclopentylidenehydrazide	0
Benzoic acid, p-nitro-, 2, 4-dichlorophenyl ester	0
Benzoic acid, p-nitro-, 2-ethylbutylidenehydrazide	0
Benzoic acid, p-nitro-, hydrazide	0
Benzoic acid, p-nitro-, isopropylidenehydrazide	0
Benzoic acid, p-nitro-, gamma-keto-alpha-methylbutylidenehydrazide	0
Benzoic acid, p-nitro-, alpha-methybenzylidenehydrazide	0
Benzoic acid, p-nitro-, methylenehydrazide	0
Benzoic acid, p-nitro-, alpha-methylpropylidenehydrazide	0
Benzoic acid, p-nitro-, p-nitrophenyl ester	0
Benzoic acid, p-nitro-, pentachlorophenyl ester	0
Benzoic acid, p-nitro-, piperonylidenehydrazide	0
Benzoic acid, p-nitro-, propylidenehydrazide	0
Benzoic acid, m-nitro-2-(2-pyridyl) ethyl ester	0
Benzoic acid, p-nitro-, 2-(2-pyridyl) ethyl ester	0
Benzoic acid, p-nitro-, tetrahydrofurfuryl ester	0
Benzoic acid, p-nitro-, m-tolyl ester	0
Benzoic acid, p-nitro-, o-tolyl ester	0
Benzoic acid, p-nitro-, p-tolyl ester	0
Benzoic acid, p-nitro-, 2, 2, 2-trichloroethylidenehydrazide	0
Benzoic acid, p-nitro-, 2, 4, 6-trichlorophenyl ester	0
Benzoic acid, piperonylidenehydrazide	0
Benzoic anhydride, p-chloro-	0
m-Benzotoluidide, 2-chloro-	0
m-Benzotoluidide, 4-chloro-	0
o-Benzotoluidide, 2-chloro-	0
o-Benzotoluidide, 4-chloro-	0
p-Benzotoluidide, 2-chloro-	0
p-Benzotoluidide, 4-chloro-	0

Table 1. (Continued)

Chemical	Injury to peach leaves [b]
m-Benzotoluidide, 2, 4-dichloro-	0
m-Benzotoluidide, 3, 4-dichloro-	0
p-Benzotoluidide, 2, 4-dichloro-	0
Benzoxazole, 2-phenoxymethyl-	0
Benzoxazole, 2-phenyl-	0
Benzylamine, N-(2, 4-dinitrophenyl)-	0
2-Butanone, 2, 4-dinitrophenylhydrazone	0
Butyraldehyde, 2, 4-dinitrophenylhydrazone	0
Butyric acid, 2-(2, 4-dinitrophenyl)hydrazide	0
Caproic acid, 2, 3, 4, 5-tetrabromo-	0
Carbonic acid, bis(p-chlorophenyl) ester	0
Carbonic acid, bis(2, 4-dichlorophenyl) ester	0
Carbonic acid, bis(2, 4-dinitrophenyl) ester	0
Carbonic acid, bis(pentachlorophenyl) ester	0
Carbonic acid, bis(2, 3, 4, 6-tetrachlorophenyl) ester	0
Carbonic acid, bis(2, 4, 6-trichlorophenyl) ester	0
Carbonic acid, di-m-tolyl ester	0
Carbonic acid, di-o-tolyl ester	0
Carbonic acid, di-p-tolyl ester	0
Chloroacetic acid, p-biphenylyl ester	0
Chloroacetic acid, diester with 2, 2'-methylenebis (4-chlorophenol)	0
Chloroacetic acid, p, p'-thiodiphenyl diester	0
o-Cresol, 4, 6-dinitro-, p-toluenesulfonate	0
o-Cresol, alpha-(hydroxyphenylimino)-	0
1, 1, 3, 3-Cyclohexanetetrapropionitrile, 2-oxo-	0
Cyclohexanone, 2, 4-dinitrophenylhydrazone	0
2-Cyclohexene-1-one, 2, 3, 4, 4, 5, 6, 6-heptachloro-	0
Cyclohexylamine, N-(2, 4-dinitrophenyl)-	0
1, 1, 3, 3-Cyclopentanetetrapropionitrile, 2-oxo-	Medium
Cyclopentanone, 2, 4-dinitrophenylhydrazone	0
Dibenzylamine, N-(2, 4-dinitrophenyl)-	Slight
Diphenylamine, 4-chloro-2-nitro-	0
Ethane, 1-(p-tert-butylphenoxy)-2-(2, 4-dinitrophenoxy)-	0
Ethane, 1, 1-dichloro-2, 2-bis(4-hydroxy-3, 5-dinitrophenyl)-	0
Ethane, 1, 1, 1-trichloro-2, 2-bis(4-chloro-3, 5-dinitrophenyl)-	0
Ethane, 1, 1, 1-trichloro-2, 2-bis(2, 5-dimethoxyphenyl)-	0
Ethane, 1, 1, 1-trichloro-2, 2-bis(p-fluorophenyl)-	0
Ethane, 1, 1, 1-trichloro-2, 2-bis(p-fluorophenyl)-(technical)	0
Ethanol, 2-(2, 4-dinitroanilino)-	0
Ethanol, 2-(2, 4-dinitrophenoxy)-	0

Table 1. (Continued)

Chemical	Injury to peach leaves [b]
Ethanol, 2, 2'-(phenylimino)di-	0
Ethanol, 2, 2'-(m-tolylimino)di-	0
Ethanol, 2-(alpha[3], alpha[3], alpha[3]-trifluoro-m-toloxy)-	0
Ether, benzyl 2, 4-dinitrophenyl	0
Ether, 2-bromo-4-tert-butylphenyl 2, 4-dinitrophenyl	0
Ether, p-bromophenyl 2, 4-dinitrophenyl	0
Ether, 4-tert-butyl-2-chlorophenyl 2, 4-dinitrophenyl	0
Ether, p-tert-butylphenyl 2, 4-dinitrophenyl	0
Ether, o-chlorophenyl 2, 4-dinitrophenyl	0
Ether, p-chlorophenyl 2, 4-dinitrophenyl	0
Ether, 2-biphenylyl 2, 4-dinitrophenyl	0
Ether, 4-biphenylyl 2, 4-dinitrophenyl	0
Ether, cyclohexyl 2, 4-dinitrophenyl	0
Ether, o-cyclohexylphenyl 2, 4-dinitrophenyl	0
Ether, 2, 4-dichlorophenyl 2, 4-dinitrophenyl	0
Ether, p(alpha, alpha-dimethylbenzyl)phenyl 2, 4-dinitrophenyl	0
Ether, 2, 4-dinitrophenyl 4-methylcyclohexyl	0
Ether, 2, 4-dinitrophenyl alpha-naphthyl	0
Ether, 2, 4-dinitrophenyl beta-naphthyl	0
Ether, 2, 4-dinitrophenyl m-nitrophenyl	0
Ether, 2, 4-dinitrophenyl o-nitrophenyl	0
Ether, 2, 4-dinitrophenyl p-nitrophenyl	0
Ether, 2, 4-dinitrophenyl 2-nitro-p-tolyl	0
Ether, 2, 4-dinitrophenyl phenethyl	0
Ether, 2, 4-dinitrophenyl phenyl	0
Ether, 2, 4-dinitrophenyl tetrahydrofurfuryl	0
Ether, 2, 4-dinitrophenyl o-tolyl	0
Ether, 2, 4-dinitrophenyl p-tolyl	0
Ether, 2, 4-dinitrophenyl 2, 4, 6-trichlorophenyl	0
Ethylenediamine, N, N'-bis(disalicylidene)-, copper salt	Severe
Ethylenediamine, N, N'-bis(disalicylidene)-, ferrous salt	0
Ethylenediamine, N, N'-bis(disalicylidene)-, nickel salt	0
Ethylenediamine, N, N'-bis(disalicylidene)-, zinc salt	0
Formaldehyde, 2-4-dinitrophenylhydrazone	0
Glycine, N-(2, 4-dinitrophenyl)-	Slight
2-Heptanone, 2, 4-dinitrophenylhydrazone	0
Hippuric acid, p-chloro-	0
Hydrazine, 1, 2-dibenzoyl-	0
Hydroquinone, diacetate	0
Hydroquinone, 2, 5-di-tert-butyl-	0
2-Imidazolidinethione	Moderate
Isobutyraldehyde, 2, 4-dinitrophenylhydrazone	0
Isobutyric acid, 2-(2, 4-dinitrophenyl) hydrazide	0
5(4)Isoxazolone, 4-cinnamylidene-3-methyl-	0

Table 1. (Continued)

Chemical	Injury to peach leaves[b]
Mesityl oxide, 2, 4-dinitrophenylhydrazone	0
Methanetripropionitrile, p-anisoyl-	0
Methanetripropionitrile, benzoyl-	Slight
Methanetripropionitrile, p-bromobenzoyl-	0
Methanetripropionitrile, p-chlorobenzoyl-	Slight
Methanetripropionitrile, 3, 4-dichlorobenzoyl-	0
Methanetripropionitrile, 2-naphthoyl-	0
Methanetripropionitrile, p-toluyl-	0
Morpholine, 4-(o-chlorobenzoyl)-	0
Morpholine, 4-(p-chlorobenzoyl)-	0
Morpholine, 4-(2, 4-dichlorobenzoyl)-	Moderate
Morpholine, 4-(3, 4-dichlorobenzoyl)-	0
Morpholine, 4-(p-nitrobenzoyl)-	0
Naphthalene, 1, 5-bis(2, 4-dinitrophenoxy)-	0
1, 5-Naphthalenediol	0
1-Naphthalenepropionamide, N-cyclohexyl-2-hydroxy-	0
1-Naphthalenepropionitrile, 2-hydroxy-	0
3-Napthoic acid, 1, 1-methylene bis [2-hydroxy-	0
1-Naphthol, acetate	Medium
2-Naphthol, acetate	Medium
5(4H)-Oxazolone, 2-phenyl-4-piperonylidene-	0
Phenetole, 2, 4-dinitro-	0
Phenol, o-(anisylideneamino)-	0
Phenol, o-(benzylideneamino)-	0
Phenol, p-butylamino-	0
Phenol, o-(o-chlorobenzylideneamino)-	0
Phenol, o-(p-chlorobenzylideneamino)-	0
Phenol, 4-chloro-2, 6-dinitro-	Very severe
Phenol, o-cyclohexyl-4, 6-dinitro-, p-toluenesulfonate	0
Phenol, o-(2, 4-dinitroanilino)-	0
Phenol, p-(2, 4-dinitroanilino)-	Slight
Phenol, 2, 4-dinitro-, p-toluenesulfonate	0
Phenol, p-methoxy-	0
Phenol, 2, 2'-methylenebis[4-chloro-, diacetate	0
Phenol, p-nitro-, acetate	0
Phenol, m-nitro-, p-toluenesulfonate	0
Phenol, 2, 2'-sulfonylbis[4-chloro-	0
Phenol, 2, 4, 6-tribromo-, acetate	0
Phenol, 1 trichloromethyl-4, 4'-methylenebis[2, 6-dichloro-	0
Pimelonitrile, gamma-benzoyl-gamma-methyl-	0
beta-Pinene, carbon tetrachloride addition product (49% Cl)	0
beta-Pinene, hexachloroethane addition product (59% Cl)	0

Table 1. (Continued)

Chemical	Injury to peach leaves[b]
Piperidine, 1-(o-chlorobenzoyl)-	0
Piperidine, 1-(p-chlorobenzoyl)-	0
Piperidine, 2, 4-dichlorobenzoyl-	0
Piperidine, 1-(p-nitrobenzoyl)-	0
Propane, 1, 3-dichloro-2, 2-bis(chloromethyl)-	0
1, 3-Propanediol, bis(chloromethyl)-	0
1, 3-Propanediol, 2, 2-bis(chloromethyl)-, bis(p-chlorobenzoate)	0
1, 3-Propanediol, 2, 2-bis(chloromethyl)-, bis(2, 4 dichlorobenzoate)	0
1-Propanol, 3-chloro-2, 2-bis(chloromethyl)-	0
1-Propanol, 3-chloro-2, 2-bis(chloromethyl)-, p-chlorobenzoate	0
1-Propanol, 3-chloro-2, 2-bis(chloromethyl)-, 2, 4-dichlorobenzoate	0
Propionic acid, 2-(2, 4-dinitrophenyl)hydrazide	0
Pyrophosphoramide, octomethyl-	0
p-Quinone, 2, 5-di-tert-butyl-	0
Sulfanilanilide, 4'-nitro-	0
Sulfide, bis(2, 4-dinitrophenyl)-	0
Sulfone, benzyl p-chlorophenyl-	0
Sulfone, bis(p-chlorophenyl)	0
Sulfone, p-chlorobenzyl p'-chlorophenyl-	0
Sulfone, p-chlorobenzyl phenyl	0
4-Thiazoline, 4-phenyl-3-o-tolyl-2-(o-tolylimino)-	Slight
Thiophosphate, O-ethyl-O, O-bis(p-nitrophenyl)-	0
Tris(p-nitrophenyl) phosphate	0
s-Trithiane	0

Table 2. Organic chemicals found possessing fungicidal properties when formulated with an equal weight of pyrophyllite.

Compound [a]	Days effective during 14-day test period [b] against conidia of		Injury to peach leaves [c]
	Monilinia fructicola	Glomerella cingulata	
Acetic acid, o-cyclohexylphenoxy-	7	7	0
Acetic acid, 2, 4-dinitrophenoxy-	2 (2)	2 (2)	Medium
Anisamide, N-ethyl-	2	2	Mild
Anisamide, N-methyl-	2	2	Medium
Anisamide, N-propyl-	7	7	Moderate
Benzamide, N-ethyl-p-nitro-	14	14	0
Benzamide, N-hydroxy-	2 (2)	2 (2)	Medium
Benzoic acid, p-chloro-, phenylhydrazide	6	6	0
Benzoic acid, 2, 4-dichloro-, 2-methyl-2-nitrophenyl ester	2 (2)	2 (2)	Medium
Benzoic acid, isopropylidenehydrazide	2 (10)	2 (10)	0
Biuret, 2, 4-dithio	2 (2)	2 (2)	Moderate
Carbonic acid, bis(p-nitrophenyl) ester	14	14	0
Ether, 2-cyclohexyl-4, 6-dinitrophenyl-2, 4 dinitrophenyl	14	14	Moderate
Ethylenediamine, N, N'-bis(disalicylidene), cobalt salt	1 (1)	1 (1)	Severe
5(4)-Isoxazolone, 4-benzylidene-3-methyl-	7	7	0
Naphthoquinone, 2, 3, 6, 7-tetrachloro-5, 6, 7, 8-tetrahydro-	14	14	0
Oxindole, 3-hydroxy-3-nitrophenyl-	2	2	0
Phenol, 2-chloro-4, 6-dinitro-	2 (2)	2 (2)	Very severe
Phenol, 2, 2'-thiobis[4-chloro-	14	14	0
p-Quinone, 2, 3, 5-trichloro-6-phenyl-	14	14	Mild
Rhodanine, 5-ethylidene-	7	7	0
Salicylamide, N-propyl-	7	7	0
p-Toluhydroquinone	2	2	0
m-Toluic acid, 2-(2, 4-dinitrophenoxy)-	2	2	0

[a] See footnote a, Table 1.
[b] Figures in parenthesis show length of test period when less than 14 days.
[c] See footnote b, Table 1.

Table 3. Fungicidal and phytotoxic properties of the most promising organic chemicals when mixed with DDT, benzenehexachloride, parathion, or pyrophyllite.

Compound[a]	Adjuvant[b]	Days effective during test period[c] against conidia of		Injury to peach foliage[d]	Rainfall during test
		Monilinia fructicola	Glomerella cingulata		
					inches
Acetic acid, o-cyclohexylphenoxy-	Pyrophyllite	7 (14)	7 (14)	0	3.16
	Benzenehexachloride	0 (14)	0 (14)	0	3.16
	DDT	3 (14)	3 (14)	0	3.16
Anisamide, N-propyl-	Pyrophyllite	7 (21)	7 (21)	Moderate	4.29
	Benzenehexachloride	7 (21)	7 (21)	Moderate	4.29
	DDT	7 (21)	7 (21)	Moderate	4.29
	Parathion	2 (21)	2 (21)	Severe	4.29
Benzamide, N-ethyl-p-nitro-	Pyrophyllite	21 (21)	21 (21)	0	0.98
	Benzenehexachloride	14 (21)	14 (21)	0	5.10
	DDT	14 (21)	14 (21)	0	5.10
	Parathion	21 (21)	21 (21)	0	5.10
Carbonic acid, bis (p-nitrophenyl) ester	Pyrophyllite	14 (14)	14 (14)	0	3.42
	Benzenehexachloride	14 (14)	14 (14)	0	3.42
	DDT	14 (14)	14 (14)	0	3.42
	Parathion	14 (14)	14 (14)	0	5.98
Ether, 2-cyclohexyl-4, 6-dinitrophenyl-2, 4-dinitrophenyl	Pyrophyllite	21 (21)	21 (21)	Moderate	4.29
	Benzenehexachloride	14 (21)	14 (21)	Severe	4.29
	DDT	21 (21)	21 (21)	Moderate	4.29
	Parathion	21 (21)	21 (21)	Severe	4.29
5(4)-Isoxazolone, 4-benzylidene-3-methyl-	Pyrophyllite	7 (14)	7 (14)	0	5.98
	Benzenehexachloride	7 (14)	7 (14)	0	5.98
	DDT	3 (14)	3 (14)	0	5.98
	Parathion	7 (14)	7 (14)	0	5.98
Naphthoquinone, 2, 3, 6, 7-tetrachloro-5, 6, 7, 8-tetrahydro-	Pyrophyllite	21 (21)	21 (21)	0	0.98
	Benzenehexachloride	14 (14)	14 (14)	0	3.42
	DDT	14 (14)	14 (14)	0	5.98
	Parathion	14 (14)	14 (14)	0	5.98
Phenol, 2, 2'-thiobis [4-chloro-	None	21 (21)	21 (21)	0	0.98
	Pyrophyllite	21 (21)	21 (21)	Mild	5.19
	DDT	28 (28)	28 (28)	0	1.76

[a] See footnote [a], Table 1.
[b] Pyrophyllite 1-100; benzenehexachloride 6% gamma 2-100; DDT 50% wettable 2-100; 15% parathion 1-100.
[c] Test period in parenthesis.
[d] See footnote [b], Table 1.

Table 3. (Continued)

Compound[a]	Adjuvant[b]	Days effective during test period[c] against conidia of *Monilinia fructicola*	Days effective during test period[c] against conidia of *Glomerella cingulata*	Injury to peach foliage[d]	Rainfall during test
					inches
p-Quinone, 2, 3, 5-trichloro-6-phenyl-	Pyrophyllite	21 (21)	21 (21)	Mild	4.29
	Benzenehexachloride	21 (21)	21 (21)	Medium	4.29
	DDT	21 (21)	21 (21)	Medium	4.29
	Parathion	21 (21)	21 (21)	Medium	4.29
Rhodanine, 5-ethylidene-	Pyrophyllite	7 (14)	7 (14)	0	5.98
	Benzenehexachloride	3 (14)	3 (14)	0	5.98
	DDT	3 (14)	3 (14)	0	5.98
Salicylamide, N-propyl-	Pyrophyllite	7 (14)	7 (14)	0	3.42
	Benzenehexachloride	4 (14)	4 (14)	0	3.42
	DDT	9 (14)	9 (14)	0	3.42
	Parathion	2 (14)	2 (14)	0	3.42

Summary

Data are presented as to the fungicidal and phytotoxic properties of 412 synthetic organic compounds. Of this total 24 compounds were found to possess fungicidal properties, but only 5 appeared promising enough to warrant further orchard tests. These are N-ethyl-*p*-nitrobenzamide; the bis(*p*-nitrophenyl) ester of carbonic acid; 2, 3, 6, 7-tetrachloro-5, 6, 7, 8-tetrahydronaphthoquinone; 2, 2'-thiobis(4-chlorophenol); and 2, 3, 5-trichloro-6-phenyl-*p*-quinone.

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 206

PLANT PATHOLOGICAL INVESTIGATION
IN THE UNITED STATES
V

Supplement 206 September 15, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

PLANT PATHOLOGICAL INVESTIGATION IN THE UNITED STATES V

Plant Disease Reporter
Supplement 206 September 15, 1951

RESEARCH IN PLANT PATHOLOGY AND BOTANY AT LOUISIANA STATE UNIVERSITY

C. W. Edgerton

The research work in botany and plant pathology in Louisiana State University and Louisiana Agricultural Experiment Station practically had its beginning when Adams Fund money became available in 1906. Previous to that date, W. R. Dodson, who later became director of the Experiment Station, made a study of the sugarcane plant and also reported on the weeds occurring in rice fields. Although at that time farmers and planters had become somewhat cognizant of such troubles as the black rot of sweetpotatoes, the cucumber mildew, and some trouble with sugarcane, practically no definite information was available in regard to the plant diseases of the State. Sugarcane planters were perhaps the first to ask for such information.

In 1906, H. R. Fulton was appointed as the first plant pathologist of the Station. During the two years that he held that position, he made some preliminary studies and surveys, and reported in bulletins on certain of the diseases of rice, peppers, and beans, and on the root rot of sugarcane.

In 1908, following the resignation of Dr. Fulton, C. W. Edgerton was appointed plant pathologist. As pathologist of the Station and later (1924-1950) as Head of the Department of Botany, Bacteriology and Plant Pathology in the University, he was in charge of the research in botany and plant pathology until his retirement in 1950. Upon his retirement, S. J. P. Chilton became Chairman of the department and is now in charge of the research work.

Starting from scratch, as would be expected, the additions to the department Staff were at first very few. C. C. Moreland was appointed as assistant in 1911 and remained on the Staff with the exception of a few years in the armed services until his resignation in 1924. The department began to expand in 1924 and this expansion has continued until the present time. Since 1924, the following have been added to the Staff in the Experiment Station: E. C. Tims (1924-date); I. L. Forbes (1925-date); E. V. Abbott (1925-26); P. J. Mills (1926-date); A. G. Plakidas (1927-date); H. H. Flor (1928-30); L. H. Person (1929-47); T. C. Ryker (1936-50); S. J. P. Chilton (1940-date); P. H. Dunckelman (1940-date); R. T. Gibbens (1940-41); W. J. Luke (1941-42); F. J. LeBeau (1944-46); J. G. Atkins (1947-date); W. J. Martin (1947-date); E. R. Stamper (1948-date).

The following men from the teaching faculty have carried on projects of their own or have assisted in various ways in the research program: C. F. Moreland, W. N. Christopher, C. A. Brown, L. H. Flint, C. S. McCleskey, L. S. Olive, Mel T. Cook, H. E. Wheeler, H. W. Johnson, and R. E. Atkinson.

The following research men from the U. S. Department of Agriculture have at times been stationed in Louisiana with offices in Louisiana State University: D. C. Neal, E. L. LeClerg, T. P. Dykstra, D. C. Bain and T. T. Ayers at Baton Rouge, and W. H. Tisdale at Crowley.

Much credit should also be extended to the numerous graduate students who have from time to time carried on projects of their own or have assisted in the Experiment Station investigations.

Louisiana Conditions and Problems

Conditions in Louisiana are extremely favorable for the development and spread of most plant diseases. The rainfall is heavy (close to 60 inches in the sugarcane belt), the humidity is relatively high, and the temperatures are usually favorable during most of the year. The winters

are comparatively mild and plant growth is seldom at a standstill during any period of the year. The number of plants grown is very large, including not only those that are adapted to sub-tropical conditions, but also most short-season crops common to northern climates which find conditions favorable for growth during the winter months. The plant pathologist finds no stopping place in his work, nor any season during which he must confine his investigations to the laboratory or the greenhouse.

During the period covered by this report, which comprises practically the first half of the 20th century, many diseases and disease problems have been under investigation. The major crops such as sugarcane, cotton, rice, vegetable crops, and strawberries have of necessity received the most attention, but the diseases of other crops have been studied as time has allowed. It has also been possible to investigate certain mycological and physiological problems. During recent years, it has been necessary to take over and push weed control problems.

Sugarcane Diseases

Sugarcane diseases have received a great deal of attention through all the years. Three diseases of major importance, red rot (Physalospora tucumanensis), root rot, and mosaic have occurred in the State, along with several others of lesser importance. These diseases have caused and are still causing heavy losses. There have been periods of declining yields and crop failures. The prosperity of the sugar industry depends to a very large extent on the efficiency of the measures that are used to control these diseases. The fact that the 1950 crop was one of the best on record shows that the methods adopted have been fairly efficient.

The red rot was first observed in the State in 1908. In the years that followed, information was obtained on the losses caused by the disease, how the fungus enters the plant, how the spores migrate through the ducts of the fibro-vascular bundles, how the cells of resistant and susceptible plants react to the invading mycelium, and on the occurrence of the perfect stage. The disease reduces the sucrose content of the juice and is also often responsible for poor stands in the field. The mosaic (virus) was recognized in 1919. This disease made it necessary to discard all the varieties that were being grown at that time. At present, with one exception, all varieties grown are resistant. Consequently, the disease has lost much of its importance. The root rot, which is considered to be caused by a complex of factors, is still not very well understood.

The breeding program which has been carried on by the U. S. Department of Agriculture and now also by the Department of Plant Pathology in the Experiment Station has been responsible for the production of canes which are fairly resistant to the major diseases. At present, varieties are not released for planting unless they are resistant.

It is not necessary at this time to present further details in regard to the sugarcane investigations. A bulletin has just been published by the Experiment Station concerning work of the department on sugarcane diseases (Edgerton, C. W. Forty-two years of sugarcane disease research at the Louisiana Agricultural Experiment Station. Louisiana Agr. Exp. Sta. Bul. 448. 1950). A copy of this bulletin may be obtained from the Director of the Experiment Station, L. S. U., Baton Rouge, Louisiana.

Cotton Diseases

In the earlier period of the work, considerable attention was given to the diseases of cotton affecting the bolls. In 1912, a bulletin on the boll rots of cotton was published. This bulletin, which included the information obtained in Louisiana, has been used as a reference in most cotton-producing countries of the world. In this bulletin, the Diplodia boll rot was for the first time listed among the more important cotton diseases. The perfect stage of the cotton anthracnose (Glomerella gossypii) was reported and data indicating that both the cotton anthracnose and the angular leaf spot organisms (Xanthomonas malvacearum) could gain entrance to the cotton bolls through the deteriorating flower parts was presented. It was also shown that the angular leaf spot lesions on the cotton bolls acted as courts of infection for the anthracnose. It was shown that the cotton anthracnose was carried over from one season to the next by spores on the surface of the seed and by mycelium within the seed. It was also shown that these spores and mycelium would die out during the first year of seed storage. By a longer period of storage, anthracnose-free seed could be obtained.

In later years, Neal carried on seed treatment tests in Louisiana. In the years when most of the seed used for planting was treated, there was very little loss in the State from anthracnose and angular leaf spot. Neal also continued work on the cotton wilt (Fusarium oxysporum f. vasinfectum) and in the last few years has been observing and studying a small outbreak of Texas

root rot (Phymatotrichum omnivorum) in the northwest corner of the State.

Diseases of Vegetable Crops

Many vegetables are grown in Louisiana both for local consumption and for shipment to northern markets. From a pathological standpoint, the ones that have received the most attention include: bean, tomato, sweetpotato, Irish potato, eggplant, onion and cucumber.

Bean Diseases. In the first decade of the 20th century, most of the bean seed used for planting came from Michigan and other northeastern States. A considerable portion of this seed was affected to a greater or less extent with anthracnose (Colletotrichum lindemuthianum). As conditions in the spring in Louisiana are very favorable for anthracnose, epidemics were common and severe. In studying the disease, it was found that the causal fungus was definitely a low-temperature organism, having an optimum temperature for growth of 22-23° C. and a maximum around 30° C. This meant that clean seed could be obtained from fields planted in August. Later, it was found that the disease did not develop in many regions in the West. In recent years, the growers have depended almost entirely on western-grown seed and the disease has practically disappeared from Louisiana fields.

The bacterial blights (Xanthomonas phaseoli, Pseudomonas phaseolicola) have also been serious and at the present time are the most serious diseases of the bean. While working on these troubles, Person found that it was possible to obtain blight-free seed from certain of the dry valleys in California. For a number of years Louisiana growers contracted for such seed. It is believed that it is possible for seedsmen to obtain bean seed free of the blights as well as of the anthracnose.

Tomato Diseases. The investigations on tomato diseases in Louisiana began about 1909. At that time, in many parts of the State, it was impossible to grow the commercial varieties then available because of tomato wilt. Wilt-resistant varieties of certain other crops, particularly cotton, had been obtained, and it was reasonable to expect that wilt-resistant varieties of tomatoes could also be procured. Many varieties were tested in the field and finally a single plant was found that was highly resistant. This plant had certain undesirable characteristics and so was crossed with the Earliana, a variety that was being extensively grown at the time. Eventually two resistant varieties were obtained and were grown on a wide scale in several of the southwestern States for a number of years and are still being grown to some extent. These varieties have also been used by geneticists in various parts of the country for breeding purposes.

In the breeding work in Louisiana, a new method of selecting for resistance was developed. Seeds were planted in sterilized soil re-infested with the wilt fungus. As susceptible plants were either killed or showed the presence of the disease, these were easily eliminated and only plants showing resistance were taken to the field. This method permitted the testing of large numbers of plants.

In the studies on tomato wilt, it was found that the optimum temperature for the growth of the fungus (Fusarium oxysporum f. lycopersici) in culture and for infection in the field was about 29° C. This explains why the tomato wilt is more severe in the Southern States where the temperatures are relatively high in the spring season.

The early blight, Alternaria solani, has also been a very important factor in Louisiana. In early spraying tests, it was found that while the disease could be controlled to a certain extent, the harvest period was also delayed.

Sweetpotato Diseases. The sweetpotato is one of the major crop plants in Louisiana. During the first 20 years of the present century, because of declining returns from sugarcane and cotton, farmers centered around Sunset and Opelousas attempted to substitute the sweetpotato and to develop a commercial sweetpotato industry. The attempt was outstandingly successful. The industry gradually enlarged, and finally that area became the leading sweetpotato center in the United States. It must be admitted that the early growers were very fortunate. The industry in its beginning developed without trouble from any serious disease. The growers used their own sweetpotatoes for planting purposes and the use of vine cuttings was a common practice. This method of propagation held the black rot in check and the other serious diseases were not introduced.

During World War II and the years immediately preceding, conditions changed. Owing to increased demands for sweetpotatoes and also a greater demand for early crops, the growers shifted to the use of draws for planting. They also shipped draws and seed potatoes in from other areas. The production of draws for sale developed into an industry of considerable size. The State Department of Agriculture attempted to protect the purchasers of roots and draws by requiring that these materials be certified. This requirement was of considerable help, but in

spite of it the major diseases of sweetpotatoes became established in all of the commercial areas.

There are at present three major diseases of sweetpotatoes in Louisiana, black rot, soil rot, and stem rot (Endoconidiophora fimbriata, Actinomyces ipomoea, Fusarium oxysporum f. batatas). The research work in the Experiment Station has largely been concerned with the first two.

The losses from black rot increased enormously during this recent period in which the industry was expanding. As the losses previous to that had been of more or less minor importance, the growers did not take the necessary precautionary measures to hold it under control. It soon became a serious trouble, not only in the field and in storage, but also on roots being shipped to market. Sweetpotatoes are usually washed before shipment. It was found that roots washed in water badly contaminated with spores of the black rot fungus would be seriously affected and sometimes become a total loss before reaching the northern markets. In tests made at the shipping centers, Martin and Person showed that this loss could be largely eliminated by dipping the roots in a weak borax solution. In recent work, results obtained by U. S. Department of Agriculture workers at Beltsville have been confirmed. It has been found that exposing black-rot-affected roots at a temperature of 43° C. for a period of 24 hours destroys the fungus without injuring the roots. Additional work here indicates that exposures of 30 hours at 40° C. also are lethal to the fungus in affected roots. This has not as yet been used commercially but the method looks promising.

The soil rot was first recognized in the vicinity of Sunset in 1934. During the years that followed, it spread rather rapidly and became a disease of considerable importance. Sweetpotato growing was abandoned on some farms. Serious trouble has developed only in dry years.

In Louisiana, as had been suggested earlier in other States, it was found that the disease was caused by an Actinomycete. The organism was isolated and tested and finally in 1940 was described as a new species, Actinomyces ipomoea, by Person and Martin.

After extensive tests, Person found that the disease could be controlled in the Sunset region by treating the fields with about 500 lbs. of sulfur per acre. The organism does not live in a soil testing pH 5.2 or lower. The soils around Sunset ordinarily test about pH 4.8, which could be reduced to about pH 5.0 by the addition of sulfur. It was found that potatoes could be grown on such treated soils for several years without additional treatment.

White Potato Diseases. In Louisiana, the Irish potato is grown largely for shipment to northern markets in the early spring. The yields are not large but prices in the spring are usually high enough to make the growing of potatoes reasonably profitable. The greatest hazards to the industry have been the occurrence of certain diseases, the most troublesome including the mosaics, ring rot, and late blight, for which climatic conditions in Louisiana are extremely favorable for development in epidemic form. Since these diseases are all seed-borne, and Louisiana growers depend almost entirely upon northern-grown potatoes for planting purposes, control depends to a great extent upon the measures taken by growers in the northern seed-producing States.

Between 1910 and 1920, yield in Louisiana dropped to a point too low for potato growing to be profitable. The cause of the decline was at first not known, but in later years was recognized to be the extremely high infection with certain mosaics, particularly those of the rugose type. Mosaics were just becoming recognized then and even potato pathologists did not appreciate their importance. Also, at that time seed certification programs were just beginning to be set up in a number of the northern States, first aimed mainly at guaranteeing purity as to variety of the seed stocks sold for planting.

In Louisiana, the idea developed that it might be possible to obtain certified seed potatoes from fields showing a minimum of mosaic, or seed stocks from regions in which the mosaic did not spread rapidly. At first, it was not possible to persuade seed certifying agencies to include a tolerance for mosaic of less than 30 to 40 percent, but it did not take long to demonstrate that this high tolerance was entirely unsatisfactory. On the advice of the Experiment Station, a large shipment of several cars of seed potatoes, said to have been obtained from a field showing about 30 percent of mosaic, was purchased and used for planting in Louisiana in 1921. The growers barely did better than to get their seed back. Some fields were not harvested. This very suddenly and very definitely settled the question in the negative as to whether seed potatoes with an appreciable amount of mosaic could be used for planting in Louisiana. Eventually the tolerance for mosaic dropped to around 2 percent. With better and more healthy seed stocks, the acreage planted to potatoes in Louisiana again increased.

The introduction of the ring rot (Corynebacterium sepedonicum) into the United States presented some problems which were temporarily disturbing. Recognizing the seriousness of the disease, the Louisiana State Department of Agriculture, on the recommendation of the pathologist of the Experiment Station, placed a zero tolerance on ring rot on certified potatoes shipped into

the State. This worked out very satisfactorily and the losses from ring rot have been relatively small.

During the past six or eight years, epidemics of late blight, some of them very destructive, have occurred in the State. These epidemics had not been expected, as it had been assumed that the late blight fungus (Phytophthora infestans) was a low-temperature organism and would not develop under normal Louisiana conditions. In studying the disease, Martin has isolated strains that show marked differences in their ability to survive exposures at 36° C. Whether the disease will continue to be an important factor in the production of potatoes in Louisiana is still not clear.

Onion Diseases. Of the various diseases which attack onions and shallots in Louisiana, the downy mildew and pink root (Peronospora destructor, Pyrenochaeta terrestris) seem to be the most important. These diseases have from time to time been responsible for declining yields and also crop failures. In the years between 1918 and 1921, a survey was made to determine what diseases were present and attempts were also made to control the mildew with sprays. Bordeaux mixture and Bordeaux with a sticker were used. The waxy surface of the leaves prevents proper covering with the sprays and the results obtained were not considered satisfactory.

In recent years, Tims has tested many fungicides and stickers with varying results. Tims has also reported on white rot (Sclerotium cepivorum) and several virus diseases which have become established in the State. He is now testing a large number of hybrids hoping that he can find one of good quality and also resistant to mildew.

Cucumber Diseases. The growing of cucumbers is an important industry in certain small areas of the State. Success in growing this crop, however, has always depended upon how well the growers have been able to control the two important diseases, downy mildew and anthracnose. Downy mildew (Pseudoperonospora cubensis) develops practically every year while anthracnose (Colletotrichum lagenarium) seems to be important only in wet years.

Bordeaux mixture has been used intermittently for many years to control the mildew. It was apparently first used shortly after information in regard to it was brought back from Europe by some of the U. S. Department of Agriculture investigators in the last decade of the 19th century. Experiments by the Louisiana Experiment Station on the control of the two diseases have been in progress for 10 years or more under the direction of Plakidas, LeBeau, and Atkins. It has been found that Bordeaux mixture will control downy mildew but that considerable injury from burning is often associated with its use. Bordeaux mixture, however, does not control anthracnose, and there have been disappointments in the past in years when the conditions were favorable for the development of this disease. In recent years, the newer fungicides have been tried and it has been found that certain of them, particularly Fermate and Dithane Z-78, will control both the mildew and the anthracnose.

Cereal Diseases

Investigations have been carried on with the diseases of two of the important cereals grown in Louisiana, rice and oats. Rice is one of the crops of major importance, being grown mainly in Southwest Louisiana, and along the Mississippi River and some of the bayous in the southern part of the State. Oats are grown to a limited extent in various parts of the State, both for winter grazing and for the production of grain.

Rice Diseases. A report on the rice diseases of Louisiana was made by Fulton as early as 1908. He found four diseases in the State: blast (Piricularia oryzae), green smut (Ustilaginoidea virens), black smut (Tilletia [Neovossia] horrida), and a specking of the grains primarily caused by an insect.

Other diseases have since been recognized in the State. The Cercospora leaf spot, which has been one of the more important, was studied by Ryker and Chilton, who were able to select varieties that were temporarily resistant. They found, however, that there were many strains of Cercospora oryzae differing in pathogenicity. Varieties selected for resistance eventually became attacked by strains which seemed to be new. About six or eight different strains of the fungus were isolated and tested.

Other diseases being studied at the present time include Sclerotium stem rot and white tip (Leptosphaeria calvinii).

Because of recommendations made by the Experiment Station, many growers are now treating their seed before planting. When conditions for growth have not been satisfactory, better stands have been obtained with treated seed.

Oat Diseases. Brief mention may be made of two of the more important oat diseases, crown rust and Helminthosporium blight. Climatic conditions in Louisiana are extremely favorable for the development and spread of crown rust (Puccinia coronata). For years, the growers were compelled to depend entirely on varieties of the Texas Red Rustproof group of oats. As a result

of the oat-breeding program, newer varieties eventually became available. These were checked closely, not only for resistance to rust, but for other desirable characters. The Victoria and Bond hybrids were found to be especially valuable. These were accepted by the growers and for a period of years were the leading varieties in the State.

In a study made of crown rust, Forbes found that the urediniospores do not live over the summer season in Louisiana. This meant that the spores responsible for the initial infections of crown rust in December and January must come from outside the State.

The Helminthosporium blight (H. victoriae) was responsible for a change in the oat program. The disease did not enter the State for two or three years after it had caused trouble in other regions. Because of the very favorable conditions in Louisiana, it seemed probable that the disease would be very troublesome. Consequently when the disease was first reported in the State, a letter was sent to all county agents and to the principal oat growers, recommending that no further plantings of the Victoria hybrids be made. Fortunately, this recommendation was very generally followed and the losses from this disease were held to a minimum. Later, studies of the disease were made by Stamper and Atkins and it was shown that there is little chance of growing the present Victoria hybrids in southern and central Louisiana.

Forage Crop Diseases

A survey was made of the sorghum plantings in the State by Bain in order to determine what diseases might be causing serious losses. At the time the survey was made there was a possibility that the crop might be used for the production of sugar as well as for forage. A new disease, apparently recently introduced, was found. The cause was a fungus described as Gloeocercospora sorghi by Bain and Edgerton. The disease has since been found in a number of States, not only on sorghum but also on corn and a few other grasses.

Fruit Diseases

Of the several fruits that are grown in Louisiana the strawberry is most important. Others of lesser importance include figs, pears, peaches, and species of Rubus and Citrus.

Strawberry Diseases. The strawberry is grown very extensively in the southwest part of the State, being the principal crop in Tangipahoa and parts of Ascension and Livingston Parishes. The industry was built on the Klondyke variety, which originated in Louisiana many years ago. For a long period there was little trouble, but eventually, as the industry expanded, Klondyke became severely attacked by leaf spot and to a lesser extent by the leaf scorch and strawberry-growing declined. To determine the possibility of control of these diseases, extensive tests were made by Plakidas in various parts of the strawberry belt and found that almost complete control could be obtained by spraying with Bordeaux mixture. As a result of these tests, spraying is practiced very generally by most growers and has very definitely kept the industry in a prosperous condition. The Bordeaux mixture apparently acts as an eradicant fungicide, the fungus fruiting structures being killed before the spores are produced. Plakidas has also made an extensive study of the fungi causing leaf spot and leaf scorch (Mycosphaerella fragariae, Diplocarpon earliana), determining how these fungi enter the strawberry leaf and cause infection.

Plakidas also made some investigation on the strawberry dwarf, a disease now known to be caused by nematodes (Aphelenchoides). As the disease seems to be of little economic importance, it has received but little attention in recent years. A new leaf spot which appeared rather suddenly was also studied. This was called the purple leaf spot and the organism causing it was described by Plakidas as a new species, Mycosphaerella louisianae.

In recent years strawberry varieties have changed and new pathological problems are developing which eventually will have to be considered if the industry is to be maintained at a high level.

Fig Diseases. Figs are grown in Louisiana mostly for home use and for sale locally. As the fruits are attacked very quickly by rot organisms after picking, they are rarely shipped to distant markets. As early as 1911, Edgerton reported on the diseases of figs then known to occur in the State, including several diseases caused by well-known organisms, such as anthracnose (Glomerella cingulata), soft rot (Rhizopus nigricans), rust (Physopella fici), leaf spot (Cercospora fici), and nematode root galls. Besides these, canker and limb blight were listed as new. Canker, which develops around the old fruit scars, is caused by a fungus described as Tuberculariа fici. The limb blight occurs on the limbs and branches and covers them with the conspicuous salmon-colored fructifications. The fungus which causes the disease is now known to be Corticium salmonicolor, although at the time it was incorrectly identified. This was one of the first reports of this tropical fungus on living trees in the United States.

In later years other fig diseases appeared, some of them apparently coming in from regions farther to the south, possibly from the tropics.

An interesting twig blight caused by the fungus Stilbum cinnabarinum (perfect stage, Megalonectria pseudotrichia) appeared on figs about 1932 and was studied by Tims. The disease caused death of the twigs, which were covered with the bright red fruiting structures. It spread rapidly and soon was widespread in all parts of south Louisiana. For some reason which has not been satisfactorily explained, the disease, after reaching its peak, gradually became less common and in a few years almost disappeared.

Leaf blights caused by Rhizoctonia-like fungi have been the most troublesome during the past 30 years. As shown by Tims, the organisms involved include the thread-blight fungus, Pellicularia koleroga, and one or more strains of Pellicularia filamentosa. Very commonly large branches are involved and sometimes the trees are killed. The thread-blight fungus maintains itself by rhizomorphs on the bark and so is difficult to control. Some control has been obtained by spraying with eradicant and protectant fungicides.

Two new leaf spots have been reported in south Louisiana in recent years. The organisms causing them were described by Tims and Olive with the following names, Cephalosporium fici and Ormathodium fici.

Pear Diseases. The pear blight (Erwinia amylovora) limits very definitely the pear varieties that can be grown in Louisiana. The varieties grown are mainly members of the resistant Chinese Sand pear group, the pineapple variety being the one usually planted. For years, this variety seemed to be free of any serious disease. About eight or ten years ago, however, the pear leaf-blight fungus, Fabraea maculata, entered the State, first appearing in the eastern part and gradually spreading westward. The Pineapple pear is extremely susceptible to this fungus and it is not uncommon to see trees practically defoliated. Plakidas found that the fungus overwinters in the dead leaves on the ground and attacks the new leaves near the ground very early in the spring. The disease can be controlled by spraying with copper fungicides or with Fermate, but very few growers follow the recommendations.

The Pineapple pear is also very susceptible to the root rot caused by Clitocybe tabescens. Many trees have been killed by this fungus. Plakidas was able to reproduce the disease by inoculating with pure cultures.

Rubus Diseases. Very few attempts have been made in Louisiana to grow species of Rubus on a commercial scale. Wild species of blackberries and dewberries grow in such profusion and the production of fruit is so great that there is little need of commercial plantings. A number of diseases attack Rubus plants in Louisiana and some of these undoubtedly would be important if blackberries or dewberries were to be grown on a commercial scale. One of these, rosette, has been investigated by Plakidas. In some other sections of the country, this disease is known as double blossom. It is characterized by the presence of numerous short branches giving a rosette appearance. The flowers are enlarged and severely distorted and fruit formation is prevented. It was found that the causal fungus had not been properly classified and it was renamed Cercosporella rubi. The disease is very interesting because of the peculiar host-parasite relationships. The mycelium does not enter the host cells but remains between the bud scales and the different flower parts, in close association with the embryonic bud elements. Infection takes place in the early spring on the young primocanes. Control was obtained by cutting back the young primocanes and by spraying with Bordeaux mixture.

Citrus Diseases. While the citrus industry in Louisiana has never been large, it has been very important in certain areas, especially along the Mississippi River below New Orleans. The Department of Plant Pathology of the Experiment Station cooperated closely with the United States Department of Agriculture and the State Department of Agriculture in the citrus canker eradication program, during which, with the exception of a small area from slightly above Buras to the mouth of the River, most of the citrus plantings in the State were destroyed. At present the disease seems to be totally eradicated. During the past year, another very serious disease has appeared in the main citrus-growing area of the State, and already has been responsible for the death of hundreds of trees grafted on sour orange stock. It seems to be identical with or at least very similar to the quick decline of California and the tristeza of Brazil. The disease has been reproduced by grafting.

Diseases of Ornamentals

During the past few years, interest in the growing of ornamentals in Louisiana, as well as in other parts of the South, has increased greatly. As a result, growers as well as nurserymen are recognizing the diseases that are present and are increasingly requesting information in regard to control measures. During recent years, the Department of Plant Pathology has been trying to

anticipate the problems that are apt to become important and has started investigations on some of them. As yet, Easter lilies, Oriental arborvitae, Pittosporum, and Camellias have received the most attention.

Easter Lily Diseases. The growing of Easter lily bulbs in southern Louisiana for shipment to northern markets has at times been an important industry and would become very important if satisfactory control measures could be found for some of the serious diseases. The virus diseases and the black scale have been the most important. Several virus diseases are present and some of them are serious enough to prevent the growing of bulbs on a commercial scale. Attempts have been made to control these troubles by roguing and by certification measures, but the results have not been satisfactory. Investigations on the black scale have been more encouraging. This disease is caused by a fungus which Plakidas has named Colletotrichum lilii. The scales are affected and the bulbs do not grow satisfactorily. Investigations by Plakidas and LeBeau have shown that it is possible to destroy the fungus by soaking the bulbs for about 48 hours in a Puratized solution.

Oriental Arborvitae Disease. For a time, the Oriental arborvitae was among the more important ornamentals planted. Very commonly, however, this plant was affected with a blight which killed the branches. In studying the disease, Plakidas found that it was caused by a fungus which he described as Cercospora thujina. He also found that the disease could be controlled by spraying with Bordeaux mixture and other copper fungicides. Because of this disease the arborvitae is being planted less and less.

Pittosporum Leafspot. A leafspot on Pittosporum appeared quite suddenly a few years ago. Plakidas studied the disease and described the organism causing it as Cercospora pittospori.

Camellia Diseases. At present in Louisiana there is more interest in Camellias than in other ornamental plants. As conditions must be just right for Camellias, troubles have developed from time to time. Plakidas has been studying the die-back, which seems to involve soil conditions as well as attack by certain fungi. He has also been investigating certain scab diseases. Some of these are caused by species of Sphaceloma (Elsinoë), and the cause of others is perhaps physiological. The flower blight, caused by a species of Sclerotinia, has also appeared in the State and at present is causing much concern.

Mycological Investigations

From time to time, certain strictly mycological problems have received considerable attention. The investigations have involved not only parasitic organisms but also many others which have seemed of particular interest or importance. Many of the fungi, on account of the short life cycle and the ease with which they can be handled, are excellent for working out certain basic, biological principles. The mycological investigations have included not only life cycle studies but also taxonomic, physiologic, and cytologic studies.

As a result of the taxonomic studies, many new organisms have been described and more information has been obtained in regard to certain groups of fungi. For example, the Louisiana species of Synchytrium have been studied by M. T. Cook over a period of several years. Much information is now available in regard to the species of this very interesting genus.

Among the more interesting physiologic and cytologic investigations, the work with antibiotic organisms and the studies made with Glomerella should be mentioned.

Antibiosis Investigations. During the whole course of the investigations covered by this report, antagonistic or antibiotic organisms living in the soil have been recognized as very important in eliminating certain pathogenic organisms and in determining the nature of the soil flora, and indirectly soil fertility. In one of the early publications on the bean anthracnose, it was shown that certain soil organisms, particularly a species of Fusarium, were important in killing out the anthracnose fungus in lesions on the seeds and on the emerging cotyledons.

Later, while the sugarcane root rot was being investigated, Tims isolated an Actinomycete which he reported in 1932 as being definitely antagonistic to the root-rot Pythium. In 1938, LeBeau reported that a species of Trichoderma could under some conditions destroy this Pythium.

In recent years, Cooper and Chilton made a survey of the Actinomycetes in the soils of the Sugar Belt that were antibiotic to the root rot Pythium. From 18 to 31 percent of the Actinomycetes isolated were definitely antibiotic to Pythium in culture.

Glomerella Investigations. Fungi of the genus Glomerella have been found to be extremely valuable for physiologic, cytologic and genetic studies. In 1912, Edgerton reported that with some of these, two strains designated plus and minus commonly occur. It was shown that when the two strains were placed in a culture plate, a dense ridge of perithecia formed on the line of contact where they grew together. In 1940 Chilton initiated genetic studies on this organism, and Lucas, Wheeler, McGahen, Ernest and Olive, have used various Glomerella strains for

genetic, physiologic and cytologic studies, and a series of papers based on these studies has been published. At present, the radioactive isotope, C^{14}, is being used by Wheeler in some of the investigations.

Weed Control Investigations

In recent years, interest in weeds and weed control has increased very rapidly in Louisiana as well as in other parts of the United States. At the present time many States, including Louisiana, have active projects on weed control. The present interest may be said to have had its beginning when it was discovered that certain weeds were very sensitive to 2, 4-D and could be killed by extremely small amounts of this chemical when applied in dusts or sprays. In Louisiana, investigations have been mainly in the Department of Botany and Plant Pathology, but with the cooperation of the Department of Agricultural Engineering.

Weed control investigations in Louisiana started when the State Legislature in 1944 made an appropriation to the Experiment Station for the eradication of the alligator weed (Alternanthera phylloxeroides), which had become a very serious pest in sugarcane fields in South Louisiana. A project was set up in the Department of Botany and Plant Pathology. At the time it was realized that it might be very difficult to find satisfactory control measures for this weed. To begin with, the various herbicides then known were tested. Fortunately, shortly after the work started, 2, 4-D became available and this chemical was immediately included in the tests then being made by Brown and Ryker. The alligator weed was found to be very sensitive to 2, 4-D and the planters were quick to follow the control recommendations made by the Department. As a result, in two to three years time, this weed ceased to be a problem in cultivated fields. At the same time, Ryker also demonstrated that a number of the important weeds in rice fields could be controlled with 2, 4-D.

As the results obtained from the preliminary investigations on weed control were so encouraging, it was only natural for the farmers and the planters in the State to insist that the weed projects be enlarged and that attempts be made to find successful control measures for some of the other serious weeds of the State. In cooperation with many of the chemical companies interested in herbicides, extensive investigations have been made on the control of a number of the important weeds, including Johnson grass and the weeds in cotton fields. Johnson grass is unquestionably the most troublesome weed in the State, and because it spreads by both rhizomes and seed, it is a very difficult plant to eradicate. Very promising results have been obtained by combining summer fallow with pre-emergence and post-emergence sprays. In 1951, many planters will use every effort to control Johnson grass in their fields. In the extensive tests in the cotton weed investigations, it has been necessary to use contact herbicides such as the oils and dinitro compounds, with some very encouraging results. In 1951, tests will be made on a large scale.

Graduate Instruction

During the past 20 years graduate instruction and graduate work in general in the Department of Botany and Plant Pathology have expanded rapidly. This expansion has been aided by a grant from the General Education Board and by the establishment of graduate fellowships by Louisiana State University. With the exception of the war years, there have been from 15 to 25 registered graduate students each year. In this period, more than 80 Master of Science degrees and 19 Doctor of Philosophy degrees have been granted. The graduate students have mostly worked very closely with the research men in the Department.

LOUISIANA STATE UNIVERSITY, BATON ROUGE

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
AGRICULTURAL RESEARCH ADMINISTRATION
UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 207

COMMON NAMES OF DISEASES OF WOODY PLANTS

Supplement 207 September 15, 1951

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

COMMON NAMES OF DISEASES OF WOODY PLANTS

F. P. Hubert

Plant Disease Reporter
Supplement 207

September 15, 1951

Only a few lists of common names for diseases of plants have been published. The most widely known work is the "List of Common Names of British Plant Diseases," which was prepared by a semiofficial committee of the British Mycological Society. Common names of plant diseases in both English and French were included by Crowell and Lavallée in their "Check List of Diseases of Economic Plants in Canada," compiled with the cooperation of the Common Names Committee of the Canadian Phytopathological Society. Some common names of plant diseases were also included in the "Index of Plant Diseases in the United States" prepared by Freeman Weiss.

In these three publications, which are in the nature of check lists, under the various host plants are listed the scientific names of the organisms known to cause diseases of these plants, the symptoms of the diseases, and the known geographical distribution. The injury caused by the attacking organism, together with the common name of the host, is frequently the accepted common name of the disease; e.g., oak wilt caused by Chalara quercina.

Possibly the principal factors that have discouraged the preparation of a comprehensive list in the past have been the wide range of hosts of certain pathogenic organisms and the varying symptoms of infection produced on different hosts by some pathogens, However, a close examination of this situation by the writer over a period of two years has revealed that a number of common names of plant diseases have been generally accepted in the field of phytopathology as the result of long usage in literature or because of their particular aptness. For example, the common name "crown gall" on its numerous hosts has, over a period of time, become established as the name of the disease caused by the bacterium Agrobacterium tumefaciens. Because of the apt description of the disease symptom, "mimosa wilt" has been accepted generally as the name of the disease caused by Fusarium oxysporum f. perniciosum.

The common names of plant diseases may be divided conveniently into definite groups. A common name may, of course, involve factors associated with several of these groups. In one group the names of geographical or political areas are included; for example, Eastern gall rust caused by Cronartium quercuum, Texas root rot by Phymatotrichum omnivorum, or Dutch elm disease by Ceratostomella ulmi. In these examples the common name is based on the country of origin of the disease or the area in which the disease was first or is now known to exist. In a second group the disease symptoms of the host caused by the attacking organism are utilized. This is well illustrated by the peach leaf curl caused by Taphrina deformans on peach trees. In the third group the scientific generic name of the pathogen or other organism causing the disease is the distinctive factor. Dothichiza canker of poplar, caused by Dothichiza populea, is an example of this type of common name. In a fourth group the common name of the pathogen or other causal organism is transferred in its entirety as the common name of the plant disease. Thus, quince rust is the common name not only of the fungus Gymnosporangium clavipes but of the resultant disease on the host plant. In a fifth group the common names of the host and the alternate host plants are used; for example, the cedar-apple rust disease caused by the fungus Gymnosporangium juniperi-virginianae, which attacks cedar and apple trees.

The format of this list of common names of diseases of woody plants is the same as that used by C. F. W. Muesebeck in "Common Names of Insects Approved by the American Association of Economic Entomologists." In part I the diseases are listed alphabetically by their common names with the scientific names of their causes placed opposite them. In part II the causes are listed by their scientific names with the common names of the diseases opposite them. When diseases have more than one common name, all are listed in part I in their respective alphabetical order, while in part II all common names are given with the scientific name of the cause.

In this list of plant diseases the geographical area is limited to the United States, Alaska, Hawaii, Panama Canal Zone, and Puerto Rico. Only diseases of woody plants of economic importance are included. Although an exhaustive search has been made to locate all such common names, no doubt some omissions have been made. Leaf spot diseases, in general, have not been included, because of their large number and often secondary or indefinite nature.

Most of the plant diseases listed are caused by fungi, but some by bacteria, viruses, nematodes, or parasitic seed plants, and one by an alga. In some of these cases the common names listed opposite the scientific names are actually the common names of the organisms. They are included in this list because these organisms are of concern in the field of phytopathology and a list would be incomplete with the omission of these parasites causing plant diseases. Common names are not included for those diseases in which the causal organisms have not been determined.

At the Seventh Annual Meeting of the American Phytopathological Society in December 1915, C. L. Shear, in his report of the committee on common names of plant diseases, recommended that certain principles be observed in compiling a list of common names of plant diseases. They include: (1) Adoption of names already in wide usage, (2) use of terms descriptive of symptoms, (3) utilization of scientific names of causal organisms, (4) use of the same name if possible for various diseases due to the same causal organism on different hosts, and (5) inclusion of bacterial and nonparasitic diseases and those of unknown origin. With the exception of diseases of unknown origin the writer has observed these principles insofar as practicable.

The names in this list do not all agree with those given by the Canadian workers Crowell and Lavellée for identical causal organisms, but the two lists have many names in agreement. There is less agreement with the list of common names of British plant diseases because of national differences in description of symptoms.

For the most part the common names for the diseases of woody plants have been selected from the common names or the symptoms found opposite causal organisms in Freeman Weiss' "Check List Revision of Diseases of Economic Plants in the United States." This exhaustive and outstanding work, issued in the Plant Disease Reporter in sections over a period of years, has been published in part as the "Index of Plant Diseases." Common names have also been drawn from the Plant Disease Reporter and from Phytopathology. The bibliography at the end of this article contains a number of additional sources from which common names were selected for the woody plant diseases in this list.

The writer has been extremely fortunate in having the helpful advice and constructive criticism of Freeman Weiss both in the preparation and finally in the review of this paper. Appreciation is also expressed to Miss Edna Buhrer, of the Division of Nematology, Bureau of Plant Industry, Soils, and Agricultural Engineering, who prepared a list of common names of nematodes for use in this paper. Acknowledgment is also made of the assistance given by the writer's co-workers in the Bureau of Entomology and Plant Quarantine.

COMMON NAMES OF DISEASES OF WOODY PLANTS

I. Diseases Listed by Common Names

Alder rust	Melampsoridium alni (Thuem.) Diet.
Aleurites bacterial leaf spot	Pseudomonas aleuritidis (McCul. & Demaree) Stapp
Amelanchier witches'-broom	Taphrina amelanchieri Mix
American hornbean leaf curl	Taphrina australis (Atk.) Gies.
American powdery mildew	Sphaerotheca mors-uvae (Schw.) Berk. & Curt.
Andromeda tar spot	Rhytisma andromedae Pers. ex Fr.
Angular leaf spot	Cercospora angulata Wint.
Apple bacterial blister spot	Pseudomonas papulans Rose
Apple black pox	Helminthosporium papulosum Berg
Apple black root rot	Xylaria mali Fromme
Apple blister canker	Nummularia discreta (Schw.) Tul.
Apple blotch	Phyllosticta solitaria Ell. & Ev.
Apple fly speck	Leptothyrium pomi (Mont. & Fr.) Sacc.
Apple frogeye leaf spot	Physalospora obtusa (Schw.) Cke.
Apple fruit fisheye rot	Corticium centrifugum (Lév.) Bres.
Apple mosaic	Marmor mali Holmes
Apple scab	Venturia inaequalis (Cke.) Wint.
Apple scurfy bark	Phyllosticta prunicola Sacc.
Apple sooty blotch	Gloeodes pomigena (Schw.) Colby
Apple spongy dry rot	Colletotrichum fructus (Stevens & Hall) Sacc.
Arbutus tar spot	Rhytisma arbuti Phill.
Arctostaphylos heart rot	Fomes arctostaphyli Long
Arizona mistletoe	Phoradendron macrophyllum Cock.
Ash bacterial canker	Pseudomonas savastanoi var. fraxini Brown
Ash white mottled heart rot	Fomes fraxinophilus (Pk.) Sacc.
Ash rust	Puccinia peridermiospora (Ell. & Tracy) Arth.
Asiatic chestnut twig blight	Cryptodiaporthe castanea (Tul.) Wehm.
Avocado scab	Sphaceloma perseae Jenkins
Azalea angular leaf spot	Septoria azaleae Vogl.

Azalea bud and twig blight	Briosia azaleae (Pk.) Dearn.
Azalea flower blight	Ovulinia azaleae Weiss
Azalea flower spot	Ovulinia azaleae Weiss
Azalea twig blight	Monilinia azaleae Honey
Bacterial angular leaf spot	Xanthomonas malvacearum (E.F.Sm.) Dowson
Bacterial blight of stone fruits	Xanthomonas pruni (E.F.Sm.) Dowson
Balsam fir butt rot	Polyporus balsameus Pk.
Banana black-tip	Helminthosporium torulosum (Syd.) Ashby
Banana freckle	Macrophoma musae (Cke.) Berl. & Vogl.
Banana Sigatoka disease	Mycosphaerella musae (Speg.) Syd.
Banana wilt	Fusarium oxysporum Schl. var. cubense (E.F.Sm.) Wr.
Barberry bacterial leaf spot	Pseudomonas berberidis Thornberry & H. W. Anders.
Beechdrops	Epifagus virginiana (L.) Bart.
Beech leaf spot	Gloeosporium fagi (Desm. & Rob.) West.
Birch leaf rust	Melampsoridium betulinum (Pers.) Kleb.
Birch powdery brown rot	Polyporus betulinus Bull. ex Fr.
Birch red leaf blister	Taphrina carnea Johans.
Birch witches'-broom	Taphrina americana Mix
Birch yellow leaf blister	Taphrina flava Farl.
Bitter rot	Glomerella cingulata (Ston.) Spauld. & Schrenk
Black locust seedling leaf blight	Fusicladium robiniae Shear
Black locust white spongy heart rot	Polyporus robiniophilus (Murr.) Lloyd
Black mold	Aspergillus niger van Tiegh.
Black or Manetti rose mold	Chalaropsis thielavioides Peyronel
Black-speckled leaf spot	Rhytisma punctatum Pers. ex Fr.
Blackberry orange rust	Gymnoconia peckiana (Howe) Trott.
Blackberry short-cycle orange rust	Kunkelia nitens (Schw.) Arth.
Bleeding canker	Phytophthora cactorum (Leb. & Cohn) Schroet.
Boxelder red stain	Fusarium reticulatum Mont. var. negundinis (Sherb) Wr.
Bramble cane gall	Agrobacterium rubi (Hildebrand) Starr & Weiss
Bramble spot anthracnose	Elsinoë veneta (Burkh.) Jenkins

Brown-checked rot	Polyporus sulphureus Bull. ex Fr.
Brown crumbly heart rot	Stereum fasciatum Schw.
Brown crumbly rot	Fomes pinicola (Sw. ex Fr.) Cke.
Brown mottled heart rot	Pholiota adiposa Fr.
Brown pocket heart rot	Fomes subrosea (Weir) Overh.
Brown rot of stone fruits	Monilinia fructicola (Wint.) Honey
Brown stringy rot	Echinodontium tinctorium (Ell.) Ell. & Ev.
Brown top rot	Fomes roseus Alb. & Schw. ex Cke.
Brown trunk rot	Fomes officinalis Vill. ex Fr.
Buckeye leaf blotch	Guignardia aesculi (Pk.) V. B. Stewart
Cacao canker and pod rot	Phytophthora palmivora Butl.
California buckeye witches'-broom	Taphrina aesculi (Patterson) Gies.
California chinquapin leaf blister	Taphrina castanopsidis (Ell. & Ev.) Jenkins
California mistletoe	Phoradendron californicum Nutt.
Camellia flower blight	Sclerotinia camelliae Hara
Camellia leaf gall	Exobasidium camelliae Shirai
Canadian hemlock-poplar rust	Melampsora abietis-canadensis Ludwig
Cancer root	Epifagus virginiana (L.) Bart.
Cane blight	Botryosphaeria ribis (Tode ex Fr.) Gross. & Dug. var. chromogena Shear et al.
Castor-bean white leaf spot	Cercospora ricinella Sacc. & Berl.
Catalpa brown butt rot	Polyporus catalpae Schrenk
Cedar-apple rust	Gymnosporangium juniperi-virginianae Schw.
Cedar blight	Phomopsis junipervora Hahn
Cenangium dieback of fir and pine	Cenangium ferruginosum Fr.
Cephalosporium elm wilt	Dothiorella ulmi Verrall & May
Ceratostomella elm wilt	Ceratostomella ulmi Buisman
Cherry banded chlorosis	Marmor pallidolimbatus Zeller & Milbrath
Cherry leaf spot, shot hole	Coccomyces hiemalis Higgins
Cherry mottle leaf	Marmor cerasi Zeller & Evans
Cherry powdery mildew	Podosphaera oxyacanthae (DC.) DBy.
Cherry rusty mottle	Marmor rubiginosum Reeves

Cherry vein-clearing mosaic	Marmor nerviclarens Zeller & Evans
Cherry witches'-broom	Taphrina cerasi (Fckl.) Sadeb.
Chestnut blight	Endothia parasitica (Murr.) P.J. & H.W. Anders.
Christmasberry scab	Fusicladium photinicola McClain
Citrus blast and black pit	Pseudomonas syringae van Hall
Citrus brown rot and gummosis	Phytophthora citrophthora (R.E. & E.H.Sm.) Leonian
Citrus canker	Xanthomonas citri (Hasse) Dowson
Citrus fruit end rot	Stemphylium citri Patterson & Charles
Citrus melanose or Phomopsis rot	Diaporthe citri Wolf
Citrus nematode	Tylenchulus semipenetrans Cobb
Citrus pink disease	Corticium salmonicolor Berk. & Br.
Citrus psorosis	Rimocortius psorosis (Fawcett) Holmes
Citrus sour rot	Oospora citri aurantii (Ferr.) Sacc. & Syd.
Clitocybe root rot	Clitocybe tabescens (Scop. ex Fr.) Bres.
Cocopalm nematode	Aphelenchoides cocophilus Cobb
Coffee anthracnose	Colletotrichum coffeanum Noack
Coffee brown or eye spot	Cercospora coffeicola Berk. & Curt.
Coffee viruela or iron spot	Omphalia flavida (Cke.) Maubl. & Rangel
Comandra blister rust	Cronartium comandrae Pk.
Common guava rust	Puccinia psidii Wint.
Common papaw leaf blotch	Phleospora asiminae Ell. & Morgan
Coniferous brown-felt blight	Herpotrichia nigra Hartig
Constricted mistletoe	Phoradendron ligatum Trel.
Coral spot	Nectria cinnabarina Tode ex Fr.
Cotoneaster leaf spot	Phyllosticta cotoneastri Allesch.
Cotton root rot	Phymatotrichum omnivorum (Shear) Dug.
Cranberry bitter rot	Glomerella cingulata (Ston.) Spauld. & Schrenk var. vaccinii Shear
Cranberry early rot or blast	Guignardia vaccinii Shear
Cranberry false blossom	Chlorogenus vaccinii Holmes
Cranberry hard rot	Monilinia oxycocci (Wor.) Honey
Cranberry "rose bloom"	Exobasidium vaccinii Wor.

Crapemyrtle leaf spot	Cercospora lythracearum Heald & Wolf
Crown gall	Agrobacterium tumefaciens (E.F.Sm. & Towns.) Conn
Currant anthracnose	Pseudopeziza ribis Kleb.
Currant black pustule	Phragmodothella ribesia (Pers. ex Fr.) Petr.
Currant collar rot	Fomes ribis (Schum. ex Fr.) Gill.
Currant nematode	Aphelenchoides ribes (Taylor) Goodey
Cypress rust	Gymnosporangium cupressi Long & Goodding
Cytospora canker of poplar	Cytospora chrysosperma Pers. ex Fr.
Cytospora canker of spurce	Cytospora kunzei Sacc.
Daphne leaf spot	Gloeosporium mezerei Cke.
Dense mistletoe	Phoradendron densum Torr.
Dogwood crown canker	Phytophthora cactorum (Leb. & Cohn) Schroet.
Dogwood spot anthracnose	Elsinoë corni Jenkins & Bitanc.
Dothichiza canker of poplar	Dothichiza populea Sacc. & Briard
Dothiorella elm wilt	Dothiorella ulmi Verrall & May
Douglas-fir dwarf-mistletoe	Arceuthobium douglasii Engelm.
Douglas-fir gall	Bacterium pseudotsugae Hansen & R. E. Sm.
Douglas-fir needle blight	Rhabdocline pseudotsugae Syd.
Douglas-fir needle cast	Adelopus gäumanni Rohde
Dutch elm disease	Ceratostomella ulmi Buisman
Eastern arborvitae leaf blight	Didymascella thujina (Durand) Maire
Eastern dwarf-mistletoe	Arceuthobium pusillum Peck
Eastern gall rust	Cronartium quercuum (Berk.) Miyabe
Eastern hemlock canker and twig blight	Dermea balsamea (Pk.) Seaver
Eastern hemlock needle blight	Didymascella tsugae (Farl.) Maire
Eastern hop hornbeam leaf curl	Taphrina virginica Sadeb.
Eastern mistletoe	Phoradendron flavescens (Pursh) Nutt.
Elm anthracnose	Gloeosporium inconspicuum Cav.
Elm black spot	Gnomonia ulmea (Schw. ex Fr.) Thuem.
Elm brown sapwood rot	Pleurotus ulmarius Fr.
Elm leaf blister	Taphrina ulmi (Fckl.) Johans.

Elm phloem-necrosis	Morsus ulmi Holmes
Elm wetwood	Erwinia nimipressuralis Carter
Endothia chestnut blight	Endothia parasitica (Murr.) P.J. & H.W. Anders.
Ephedra rust witches'-broom	Peridermium ephedrae Cke.
Euonymus powdery mildew	Oidium euonymi-japonici (Arcang.) Sacc.
European brown rot	Monilinia laxa (Aderh. & Ruhl.) Honey
European canker of poplar	Dothichiza populea Sacc. & Briard
European larch canker	Dasyscypha willkommii (Hartig) Rehm
European Nectria canker	Nectria galligena Bres.
European powdery mildew	Microsphaera grossulariae [Wallr.] Lév.
Feather rot	Poria subacida (Pk.) Sacc.
Fig fruit canker	Macrophoma fici Alm. & Cam.
Fig leaf spot	Cercospora fici Heald & Wolf
Fig mosaic	Marmor caricae (Condit & Horne) Holmes
Fig rust	Physopella fici (Cast.) Arth.
Filbert bacterial blight	Xanthomonas corylina P. W. Miller et al.
Fir brown pocket rot	Hydnum abietis Hubert
Fir dwarf-mistletoe	Arceuthobium campylopodum forma abietinum (Engelm.) Gill
Fir fireweed rust	Pucciniastrum pustulatum (Pers.) Diet.
Fir-huckleberry rust	Pucciniastrum goeppertianum (Kuehn) Kleb.
Fir mistletoe	Phoradendron pauciflorum Torr.
Fir needle blight	Hypodermella abietis-concoloris (Mayr) Dearn.
Fir pitted sapwood rot	Polyporus abietinus Dicks. ex Fr.
Fir twig and branch canker	Scleroderris abieticola Zeller & Goodding
Fir twig canker	Phoma abietina Hartig
Fir-willow rust	Melampsora abieti-capraearum Tub.
Fire blight	Erwinia amylovora (Burr.) Winslow et al.
Flowering-cherry rough bark	Rimocortius kwanzani Milbrath & Zeller
Gardenia bacterial leaf spot	Pseudomonas gardeniae Burkh. & Pirone
Gardenia canker and stem gall	Phomopsis gardeniae Hansen & Barrett
Grape black rot	Guignardia bidwellii (Ell.) Viala & Ravaz

Grape dead-arm, branch necrosis	Cryptosporella viticola Shear
Grape downy mildew	Plasmopara viticola (Berk. & Curt.) Berl. & de Toni
Grape powdery mildew	Uncinula necator (Schw.) Burr.
Grape spot anthracnose	Elsinoë ampelina (DBy.) Shear
Gray mold	Botrytis cinerea Fr.
Green scurf	Cephaleuros virescens O. Kunze
Hackberry downy mildew	Pseudoperonospora celtidis (Waite) G. W. Wils.
Hairy mistletoe	Phoradendron villosum Nutt.
Hairy root	Agrobacterium rhizogenes (Riker et al.) Conn
Hawthorn rust	Gymnosporangium globosum Farl.
Hazelnut canker	Apioporthe anomala (Pk.) Hoehn.
Hazelnut leaf blister	Taphrina coryli Nishida
Heath rust	Pucciniastrum ericae (Naumann) Cummins
Hevea birdseye spot	Helminthosporium heveae Petch
Hevea leaf blight	Dothidella ulei P. Henn.
Hibiscus dieback	Colletotrichum hibisci Pollaci
Hibiscus rust	Kuehneola malvicola (Speg.) Arth.
Hickory anthracnose	Gnomonia caryae Wolf
Hickory leaf blight	Septoria caryae Ell. & Ev.
Hickory twig canker	Rosellinia caryae Bonar
Hollyhock rust	Puccinia malvacearum Mont.
Honeylocust tar spot	Linospora gleditsiae Miller & Wolf
Honeysuckle leaf blight	Herpobasidium deformans Gould
Hornbeam branch and trunk canker	Pezicula carpinea (Pers. ex Fr.) Tul.
Horsechestnut leaf blotch	Guignardia aesculi (Pk.) V. B. Stewart
Horsechestnut powdery mildew	Uncinula flexuosa Pk.
Hydrangea rust	Pucciniastrum hydrangeae (Berk. & Curt.) Arth.
Incense cedar mistletoe	Phoradendron juniperinum Engelm. var. libocedri Engelm.
Incense cedar rust	Gymnosporangium libocedri P. Henn.
Juniper mistletoe	Phoradendron juniperinum Engelm.

Juniper needle blight	Chloroscypha juniperina (Ell.) Seaver
Juniper needle cast	Lophodermium juniperinum (Fr.) DeNot.
Juniper nursery blight	Phomopsis juniperovora Hahn
Juniper yellow pocket rot	Fomes juniperinus (Schrenk) Sacc. & Syd.
Larch dwarf-mistletoe	Arceuthobium campylopodum forma laricis (Engelm.) Gill
Larch needle and shoot blight	Hypodermella laricis Tub.
Larch-willow rust	Melampsora bigelowii Thuem.
Ledum spot anthracnose	Elsinoë ledi (Pk.) Zeller
Lilac bacterial blight	Pseudomonas syringae van Hall
Lilac shoot blight	Phytophthora cactorum (Leb. & Cohn) Schroet.
Lime anthracnose and withertip	Gloeosporium limetticolum Clausen
Linden anthracnose	Gnomonia tiliae Kleb.
Linden leaf blight	Cercospora microsora Sacc.
Linden powdery mildew	Uncinula clintonii Pk.
Lippia spot anthracnose	Sphaceloma lippiae Baines & Cummins
Little peach	Chlorogenus persicae var. micropersica Holmes
Locust witches'-broom	Chlorogenus robiniae Holmes
Lodgepole pine blister rust	Cronartium coleosporioides (Diet. & Holw.) Arth.
Lodgepole pine dwarf-mistletoe	Arceuthobium americanum Nutt.
Loganberry dwarf	Nanus loganobacci Holmes
London plane blight	Endoconidiophora fimbriata (Ell. & Hals.) Davidson
London plane canker stain	Endoconidiophora fimbriata (Ell. & Hals.) Davidson
Long-spiked mistletoe	Phoradendron longispicum Trel.
Loquat scab	Fusicladium eriobotryae (Cav.) Sacc.
Magnolia large leaf spot	Phyllosticta magnoliae Sacc.
Mango spot anthracnose	Elsinoë mangiferae Bitanc. & Jenkins
Maple bacterial leaf spot	Pseudomonas aceris (Ark.) Starr & Burkh.
Maple bark canker	Dermea acerina (Pk.) Rehm
Maple bark patch	Aleurodiscus acerinus (Pers. ex Fr.) Hoehn. & Litsch.
Maple black leaf blister	Taphrina dearnessii Jenkins
Maple brown leaf blister	Taphrina sacchari Jenkins

Maple tar spot	Rhytisma acerinum Pers. ex Fr.
Meadow nematodes	Pratylenchus spp.
Mesquite mistletoe	Phoradendron coloradense Trel.
Mesquite powdery mildew	Uncinula prosopodis Speg.
Mimosa wilt	Fusarium oxysporum f. perniciosum (Hepting) Snyder & Hansen
Mountain-laurel leaf blotch	Phomopsis kalmiae Enlows
Mulberry bacterial spot-blight	Pseudomonas mori Boyer & F. Lambert
Mulberry twig blight and canker	Fusarium lateritium Nees var. mori Desm.
Nectria beech bark canker	Nectria coccinea var. faginata Lohman, Watson, & Ayers
Northwestern apple anthracnose	Neofabraea malicortis Jacks.
Oak anthracnose	Gnomonia quercina Kleb.
Oak bark patch	Aleruodiscus oakesii (Berk. & Curt.) Cke.
Oak black spot	Trabutia erythrospora (Berk. & Curt.) Theiss. & Syd.
Oak brown mildew	Sphaerotheca lanestris Harkn.
Oak heart rot	Daedalea quercina L. ex Fr.
Oak leaf blsiter	Taphrina caerulescens (Desm. & Mont.) Tul.
Oak powdery mildew	Erysiphe trina Harkn.
Oak twig canker	Diplodia longispora Cke. & Ell.
Oak white root rot	Polyporus dryadeus Pers. ex Fr.
Oak wilt	Chalara quercina Henry
Ocean-spray witches'-broom	Nanus holodisci Holmes
Oleander bacterial knot	Pseudomonas tonelliana (Ferr.) Burkh.
Olive bacterial knot	Pseudomonas savastanoi (E.F.Sm.) F. L. Stevens.
Olive peacock spot	Cycloconium oleaginum Cast.
Omnivorous root rot	Phymatotrichum omnivorum (Shear) Dug.
Osage-orange cottony leaf spot	Ovularia maclurae Ell. & Langl.
Ozonium root rot	Phymatotrichum omnivorum (Shear) Dug.
Palm bud rot	Phytophthora palmivora Butl.
Palm false smut	Graphiola phoenicis Pat.
Palmetto brown felt	Septobasidium sabalis Couch

Panama disease of banana	Fusarium oxysporum Schl. var. cubense (E.F.Sm.) Wr.
Papaya fruit black spot	Ascochyta caricae Pat.
Peach asteroid spot	Marmor astri Holmes
Peach brown rot	Monilinia fructicola (Wint.) Honey
Peach frosty mildew	Mycosphaerella persica Higgins & Wolf
Peach leaf curl	Taphrina deformans (Berk.) Tul.
Peach mosaic	Marmor persicae Holmes
Peach rosette	Carpophthora rosettae Holmes
Peach scab	Cladosporium carpophilum Thuem.
Peach shoot blight	Coryneum carpophilum (Lév.) Jauch
Peach stem canker	Phoma persicae Sacc.
Peach wart	Galla verrucae Blodgett
Peach X-disease	Carpophthora lacerans Holmes
Peach yellow-red virosis	Carpophthora lacerans Holmes
Peach yellows	Chlorogenus persicae Holmes
Pear scab	Venturia pirina Aderh.
Pear stony pit	Rimocortius pyri Holmes
Pecan downy spot	Mycosphaerella caryigena Demaree & Cole
Pecan liver spot	Gnomonia caryae Wolf var. pecanae Cole
Pecan scab	Cladosporium effusum (Wint.) Demaree
Pecan spot anthracnose	Elsinoë randii Jenkins & Bitanc.
Perennial canker	Neofabrea perennans Kienholz
Persimmon wilt	Cephalosporium diospyri Crandall
Phony peach	Nanus mirabilis Holmes
Pierce's disease of grape	Morsus suffodiens Holmes
Pine brown spot of needles	Systremma acicola (Dearn.) Wolf & Barbour
Pine-oak gall rust	Cronartium quercuum (Berk.) Miyabe
Pine "pruning twig blight"	Cenangium ferruginosum Fr.
Pink wood stain	Torula ligniperda (Willk.) Sacc.
Piñon blister rust	Cronartium occidentale (Hedgc.) Bethel & Hunt
Piñon dwarf-mistletoe	Arceuthobium campylopodum forma divaricatum (Engelm.) Gill

Pittosporum angular leaf spot	Cercospora pittospori Plakidas
Plum pockets	Taphrina pruni (Fckl.) Tul.
Poinsettia canker	Corynebacterium poinsettiae Starr & Pirone
Poinsettia spot anthracnose	Sphaceloma poinsettiae Jenkins & Ruehle
Pome fruit spot anthracnose	Elsinoë piri (Woron.) Jenkins
Poplar branch and trunk canker	Dothichiza populea Sacc. & Briard
Poplar scab or shoot blight	Didymosphaeria populina Vuill.
Poplar yellow leaf blister	Taphrina populina Fr.
Prune dwarf	Nanus pruni Holmes
Prunus black knot	Dibotryon morbosum (Schw.) Theiss. & Syd.
Prunus line-pattern mosaic	Marmor lineopictum Cation
Pyracantha scab	Fusicladium pyracanthae (Otth) Rostr.
Quince fruit pale rot	Phoma cydoniae Sacc. & Schulz.
Quince rust	Gymnosporangium clavipes Cke. & Pk.
Raspberry decline	Corium ruborum Holmes
Raspberry late leaf rust	Pucciniastrum americanum (Farl.) Arth.
Raspberry leaf curl	Corium rubi Holmes
Raspberry spur blight	Didymella applanata (Niessl) Sacc.
Raspberry streak	Nanus orientalis Holmes
Red-brown butt rot	Polyporus schweinitzii Fr.
Red heart rot	Stereum sanguinolentum Alb. & Schw. ex Fr.
Red leaf gall	Synchytrium vaccinii Thomas
Red-raspberry mosaic	Marmor rubi Holmes
Red ray rot	Polyporus anceps Pk.
Red ring disease nematode	Aphelenchoides cocophilus Cobb
Red ring rot	Fomes pini (Brot. ex Fr.) Karst.
Redwood brown pocket heart rot	Poria sequoiae Bonar
Rhizina root rot	Rhizina inflata (Schaeff. ex Fr.) Sacc. (= R. undulata Fr.)
Rhododendron gray blight	Pestalotia macrotricha Kleb.
Rhododendron leaf and stem gall	Exobasidium vaccinii Wor.

Rhododendron yellow leaf spot	Exobasidium burtii Zeller
Ribes cane knot	Thyronectria berolinensis (Sacc.) Seaver
Ribes downy mildew	Plasmopara ribicola Schroet.
Rocky Mountain yellow pine dwarf-mistletoe	Arceuthobium vaginatum forma cryptopodum (Engelm.) Gill
Root knot nematodes	Meloidogyne spp. (Syn. Heterodera marioni (Cornu) Goodey)
Rose black spot	Diplocarpon rosae Wolf
Rose brand canker	Coniothyrium wernsdorffiae Laubert
Rose brown canker	Cryptosporella umbrina (Jenkins) Jenkins & Wehm
Rose graft canker	Leptosphaeria coniothyrium (Fckl.) Sacc.
Rose mosaic	Marmor rosae Holmes
Rose powdery mildew	Sphaerotheca humuli (DC.) Burr.
Rose spot anthracnose	Sphaceloma rosarum (Pass.) Jenkins
Rose streak	Marmor veneniferum Holmes
Rubus downy mildew	Peronospora rubi Rabh.
Rubus rosette and double blossom	Cercosporella rubi (Wint.) Plakidas
Rubus yellow-rust	Kuehneola uredinis (Lk.) Arth.
Sequoia trunk rot	Ganoderma sequoiae Murr.
Shoestring root rot	Armillaria mellea Vahl ex Fr.
Silver leaf	Stereum purpureum Pers. ex Fr.
Small brown dwarf-mistletoe	Arceuthobium campylopodum forma cyanocarpum (A. Nelson) Gill
Smothering of seedlings	Thelephora terrestris Ehrh. ex Fr.
Snow blight of conifer seedlings	Phacidium infestans Karst.
Snowberry spot anthracnose, scab	Sphaceloma symphoricarpi Barrus & Horsfall
Sour orange scab	Elsinoë fawcetti Bitanc. & Jenkins
Southern fusiform rust	Cronartium fusiforme (Pk.) Hedgc. & Hunt
Southern wilt	Sclerotium rolfsii Sacc.
Southwestern yellow pine dwarf-mistletoe	Arceuthobium vaginatum J. Presl
Sphaeropsis canker of elm	Sphaeropsis ulmicola Ell. & Ev.
Spongy sap rot	Fomes annosus (Fr.) Cke.

Spruce tar spot needle cast	Lophodermium piceae (Fckl.) Hoehn.
Stem and bulb nematodes	Ditylenchus spp.
Stem rust	Puccinia graminis Pers.
Stone fruit rust	Tranzschelia pruni-spinosae (Pers.) Diet.
Strawberry spring dwarf nematode	Aphelenchoides fragariae Ritz.-Bos
Strumella canker of chestnut and oak	Strumella coryneoidea Sacc. & Wint.
Sumac dieback and canker	Cryptodiaporthe aculeans (Schw.) Wehm.
Sweetfern blister rust	Cronartium comptoniae Arth.
Sweetgum red leaf spot	Leptothyriella liquidambaris Tehon & Stout
Sycamore anthracnose	Gnomonia platani (Sacc. & Speg.) Kleb.
Texas mistletoe	Phoradendron engelmanni Trel.
Texas root rot	Phymatotrichum omnivorum (Shear) Dug.
Thread blight	Pellicularia koleroga Cke.
Tip blight of conifers	Pestalotia funerea Desm.
Tree powdery mildew	Phyllactinia corylea Pers. ex Karst.
Tupelo rust	Aplopsora nyssae Ell. & Tracy
Turkish hazelnut bacterial leaf spot	Pseudomonas colurnae (Thornberry & H.W. Anders.) Burkh.
Ustulina butt rot	Ustulina vulgaris Tul.
Vaccinium blotch rot	Acanthorhynchus vaccinii Shear
Vaccinium powdery mildew	Microsphaera alni DC. ex Wint. var. vaccinii (Schw.) Salm.
Vaccinium "rose-bloom"	Exobasidium vaccinii Wor.
Vaccinium tar spot	Rhytisma vaccinii Schw. ex Fr.
Vaccinium yellow leaf spot	Exobasidium burtii Zeller
Verticillium wilt	Verticillium albo-atrum Reinke & Berth.
Viburnum bacterial leaf spot	Pseudomonas viburni (Thornberry & H.W. Anders.) Stapp
Viburnum downy mildew	Plasmopara viburni Pk.
Violet root rot	Helicobasidium purpureum Pat.
Walnut bacterial blight	Xanthomonas juglandis (Pierce) Dowson
Walnut canker and dieback	Melanconis juglandis (Ell. & Ev.) Graves

Walnut downy spot	Microstroma juglandis (Bereng.) Sacc.
Walnut leaf blight	Cylindrosporium juglandis Wolf
Walnut ring spot	Ascochyta juglandis Boltshauser
Web blight	Pellicularia filamentosa (Pat.) Rogers
Western gall rust	Cronartium harknessii Meinecke
Western hard pine dwarf-mistletoe	Arceuthobium campylopodum Engelm. forma typicum Gill
Western hemlock dwarf-mistletoe	Arceuthobium campylopodum Engelm. forma tsugensis (Rosendahl) Gill
Western maple leaf blister	Taphrina bartholomaei Mix
Western spruce dwarf-mistletoe	Arceuthobium campylopodum forma microcarpum (Engelm.) Gill
Western white pines dwarf-mistletoe	Arceuthobium campylopodum forma blumeri (A. Nelson) Gill
White flaky sapwood rot	Pleurotus ostreatus Fr.
White mottled butt rot	Fomes applanatus (Pers. ex Fr.) Gill.
White mottled wound rot	Daedalea confragosa Bolt. ex Fr.
White pine blister rust	Cronartium ribicola Ed. Fischer
White pocket rot	Polyporus pargamenus Fr.
White spongy heart rot	Polyporus hispidus Bull. ex Fr.
White spongy rot	Fomes connatus (Weinm. ex Fr.) Gill.
Willow black canker	Physalospora miyabeana Fukushi
Willow powdery mildew	Uncinula salicis DC. ex Wint.
Willow scab	Fusicladium saliciperdum (Allesch. & Tub.) Tub.
Willow tar spot	Rhytisma salicinum Pers. ex Fr.
Willow twig and branch cnaker	Cryptodiaporthe salicina (Curr.) Wehm.
Yellowish flaky heart rot	Fomes everhartii (Ell. & Gall.) Schrenk
Yellowish mottled heart rot	Fomes fomentarius (L. ex Fr.) Kickx.
Yellow leaf blister	Taphrina flava Farl.
Yellow witches'-broom	Melampsorella cerastii (Pers.) Schroet.
Yew needle blight	Sphaerulina taxi (Cke.) Mass.

II. Causal Organisms Listed by Scientific Names

Acanthorhyncus vaccinii Shear	Vaccinium blotch rot
Adelopus gäumanni Rohde	Douglas-fir needle cast
Agrobacterium rhizogenes (Riker et al.) Conn	Hairy root
Agrobacterium rubi (Hildebrand) Starr & Weiss	Bramble cane gall
Agrobacterium tumefaciens (E.F.Sm. & Town.) Conn	Crown gall
Aleurodiscus acerinus (Pers. ex Fr.) Hoehn. & Litsch.	Maple bark patch
Aleurodiscus oakesii (Berk. & Curt.) Cke.	Oak bark patch
Aphelenchoides cocophilus Cobb	Coco palm nematode Red ring disease nematode
Aphelenchoides fragariae Ritz.-Bos	Strawberry spring dwarf nematode
Aphelenchoides ribes (Taylor) Goodey	Currant nematode
Apioporthe anomala (Pk.) Hoehn.	Hazelnut canker
Aplopsora nyssae Ell. & Tracy	Tupelo rust
Arceuthobium americanum Nutt	Lodgepole pine dwarf-mistletoe
Arceuthobium campylopodum Engelm. forma abietinum (Engelm.) Gill	Fir dwarf-mistletoe
Arceuthobium campylopodum forma blumeri (A. Nelson) Gill	Western white pines dwarf-mistletoe
Arceuthobium campylopodum forma cyanocarpum (A. Nelson) Gill	Small brown dwarf-mistletoe
Arceuthobium campylopodum forma divaricatum (Engelm.) Gill	Piñon dwarf-mistletoe
Arceuthobium campylopodum forma laricis (Engelm.) Gill	Larch dwarf-mistletoe
Arceuthobium campylopodum forma microcarpum (Engelm.) Gill	Western spruce dwarf-mistletoe
Arceuthobium campylopodum forma tsugensis (Rosendahl) Gill	Western hemlock dwarf-mistletoe
Arceuthobium campylopodum forma typicum Gill	Western hard pine dwarf-mistletoe
Arceuthobium douglasii Engelm.	Douglas-fir dwarf-mistletoe
Arceuthobium pusillum Peck	Eastern dwarf-mistletoe
Arceuthobium vaginatum J. Presl	Southwestern yellow pine dwarf-mistletoe

Arceuthobium vaginatum forma cryptopodum (Engelm.) Gill	Rocky Mountain yellow pine dwarf-mistletoe
Armillaria mellea Vahl ex Fr.	Shoestring root rot
Ascochyta caricae Pat.	Papaya fruit black spot
Ascochyta juglandis Boltshauser	Walnut ring spot
Aspergillus niger van Tiegh.	Black mold
Bacterium pseudotsugae Hansen & R. E. Sm.	Douglas-fir gall
Botryosphaeria ribis (Tode ex Fr.) Gross. & Dug. var. chromogena Shear et al.	Cane blight
Botrytis cinerea Fr.	Gray mold
Briosia azaleae (Pk.) Dearn.	Azalea bud and twig blight
Carpophthora lacerans Holmes	Peach X-disease Peach yellow-red virosis
Carpophthora rosettae Holmes	Peach rosette
Cenangium ferruginosum Fr.	Cenangium dieback of fir and pine Pine "pruning twig blight"
Cephaleuros virescens O. Kunze	Green scurf
Cephalosporium diospyri Crandall	Persimmon wilt
Ceratostomella ulmi Buisman	Dutch elm disease Ceratostomella elm wilt
Cercospora angulata Wint.	Angular leaf spot
Cercospora coffeicola Berk. & Curt.	Coffee brown or eye spot
Cercospora fici Heald & Wolf	Fig leaf spot
Cercospora lythracearum Heald & Wolf	Crapemyrtle leaf spot
Cercospora microsora Sacc.	Linden leaf blight
Cercospora pittospori Plakidas	Pittosporum angular leaf spot
Cercospora ricinella Sacc. & Berl.	Castor-bean white leaf spot
Cercosporella rubi (Wint.) Plakidas	Rubus rosette and double blossom
Chalara quercina Henry	Oak wilt
Chalaropsis thielavioides Peyronel	Black or Manetti rose mold
Chlorogenus persicae Holmes	Peach yellows
Chlorogenus persicae var. micropersica Holmes	Little peach
Chlorogenus robiniae Holmes	Locust witches'-broom

Chlorogenus vaccinii Holmes	Cranberry false blossom
Chloroscypha juniperina (Ell.) Seaver	Juniper needle blight
Cladosporium carpophilum Thuem.	Peach scab
Cladosporium effusum (Wint.) Demaree	Pecan scab
Clitocybe tabescens (Scop. ex Fr.) Bres.	Clitocybe root rot
Coccomyces hiemalis Higgins	Cherry leaf spot, shot hole
Colletotrichum coffeanum Noack	Coffee anthracnose
Colletotrichum fructus (F. L. Stevens & Hall) Sacc.	Apple spongy dry rot
Colletotrichum hibisci Pollaci	Hibiscus dieback
Coniothryium wernsdorffiae Laubert	Rose brand canker
Corium rubi Holmes	Raspberry leaf curl
Corium ruborum Holmes	Raspberry decline
Corticium centrifugum (Lév.) Bres.	Apple fruit fisheye rot
Corticium salmonicolor Berk. & Br.	Citrus pink disease
Corynebacterium poinsettiae Starr & Pirone	Poinsettia canker
Coryneum carpophilum (Lév.) Jauch	Peach shoot blight
Cronartium coleosporioides (Diet. & Holw.) Arth.	Lodgepole pine blister rust
Cronartium comandrae Pk.	Comandra blister rust
Cronartium comptoniae Arth.	Sweetfern blister rust
Cronartium fusiforme (Pk.) Hedgc. & Hunt	Southern fusiform rust
Cronartium harknessii Meinecke	Western gall rust
Cronartium occidentale (Hedgc.) Bethel & Hunt	Piñon blister rust
Cronartium quercuum (Berk.) Miyabe	Eastern gall rust Pine-oak gall rust
Cronartium ribicola Ed. Fischer	White pine blister rust
Cryptodiaporthe aculeans (Schw.) Wehm.	Sumac canker and dieback
Cryptodiaporthe castanea (Tul.) Wehm.	Asiatic chestnut twig canker
Cryptodiaporthe salicina (Curr.) Wehm.	Willow twig and branch canker
Cryptosporella umbrina (Jenkins) Jenkins & Wehm.	Rose brown canker
Cryptosporella viticola Shear	Grape dead-arm, branch necrosis
Cycloconium oleaginum Cast.	Olive peacock spot
Cylindrosporium juglandis Wolf	Walnut leaf blight

Cytospora chrysosperma Pers. ex Fr.	Cytospora canker of poplar
Cytospora kunzei Sacc.	Cytospora canker of spruce
Daedalea confragosa Bolt. ex Fr.	White mottled wound rot
Daedalea quercina L. ex Fr.	Oak heart rot
Dasyscypha willkommii (Hartig) Rehm	European larch canker
Dermea acerina (Pk.) Rehm	Maple bark canker
Dermea balsamea (Pk.) Seaver	Eastern hemlock canker and twig blight
Diaporthe citri Wolf	Citrus melanose or Phomopsis rot
Dibotryon morbosum (Schw.) Theiss. & Syd.	Prunus black knot
Didymascella thujina (Durand) Maire	Eastern arborvitae leaf blight
Didymascella tsugae (Farl.) Maire	Eastern hemlock needle blight
Didymella applanata (Niessl) Sacc.	Raspberry spur blight
Didymosphaeria populina Vuill.	Poplar scab or shoot blight
Diplocarpon rosae Wolf	Rose black spot
Diplodia longispora Cke. & Ell.	Oak twig canker
Ditylenchus spp.	Stem and bulb nematodes
Dothichiza populea Sacc. & Briard	Poplar branch and trunk canker Dothichiza canker of poplar European canker of poplar
Dothidella ulei P. Henn.	Hevea leaf blight
Dothiorella ulmi Verrall & May	Dothiorella elm wilt Cephalosporium elm wilt
Echinodontium tinctorium (Ell.) Ell. & Ev.	Brown stringy rot
Elsinoë ampelina (DBy.) Shear	Grape spot anthracnose
Elsinoë corni Jenkins & Bitanc.	Dogwood spot anthracnose
Elsinoë fawcetti Bitanc. & Jenkins	Sour orange scab
Elsinoë ledi (Pk.) Zeller	Ledum spot anthracnose
Elsinoë mangiferae Bitanc. & Jenkins	Mango spot anthracnose
Elsinoë piri (Woron.) Jenkins	Pome fruit spot anthracnose
Elsinoë randii Jenkins & Bitanc.	Pecan spot anthracnose
Elsinoë veneta (Burkh.) Jenkins	Bramble spot anthracnose
Endoconidiophora fimbriata (Ell. & Hals.) Davidson	London plane canker stain London plane blight

Endothia parasitica (Murr.) P.J. & H.W. Anders.	Chestnut blight Endothia chestnut blight
Epifagus virginiana (L.) Bart.	Beechdrops Cancer root
Erwinia amylovora (Burr.) Winslow et al.	Fire blight
Erwinia nimipressuralis Carter	Elm wetwood
Erysiphe trina Harkn.	Oak powdery mildew
Exobasidium burtii Zeller	Rhododendron yellow leaf spot Vaccinium yellow leaf spot
Exobasidium camelliae Shirai	Camellia leaf gall
Exobasidium vaccinii Wor	Cranberry "rose bloom" Rhododendron leaf and stem gall Vaccinium "rose bloom"
Fomes annosus (Fr.) Cke.	Spongy sap rot
Fomes applanatus (Pers. ex Fr.) Gill.	White mottled butt rot
Fomes arctostaphyli Long	Arctostaphylos heart rot
Fomes connatus (Weinm. ex Fr.) Gill.	White spongy rot
Fomes everhartii (Ell. & Gall.) Schrenk	Yellowish flaky heart rot
Fomes fomentarius (L. ex Fr.) Kickx.	Yellowish mottled heart rot
Fomes fraxinophilus (Pk.) Sacc.	Ash white mottled heart rot
Fomes juniperinus (Schrenk) Sacc. & Syd.	Juniper yellow pocket rot
Fomes officinalis Vill. ex Fr.	Brown trunk rot
Fomes pini (Brot. ex Fr.) Karst.	Red ring rot
Fomes pinicola (Sw. ex Fr.) Cke.	Brown crumbly rot
Fomes ribis (Schum. ex Fr.) Gill.	Currant collar rot
Fomes roseus Alb. & Schw. ex Cke.	Brown top rot
Fomes subrosea (Weir) Overh.	Brown pocket heart rot
Fusarium lateritium Nees var. mori Desm.	Mulberry twig blight and canker
Fusarium oxysporum Schl. var. cubense (E.F.Sm.) Wr.	Panama disease of banana Banana wilt
Fusarium oxysporum f. perniciosum (Hepting) Snyder & Hansen	Mimosa wilt
Fusarium reticulatum Mont. var. negundinis (Sherb.) Wr.	Boxelder red stain
Fusicladium eriobotryae (Cav.) Sacc.	Loquat scab

Fusicladium photinicola McClain	Christmasberry scab
Fusicladium pyracanthae (Otth) Rostr.	Pyracantha scab
Fusicladium robiniae Shear	Black locust seedling leaf blight
Fusicladium saliciperdum (Allesch. & Tub.) Tub.	Willow scab
Galla verrucae Blodgett	Peach wart
Ganoderma sequoiae Murr.	Sequoia trunk rot
Gloeodes pomigena (Schw.) Colby	Apple sooty blotch
Gloeosporium fagi (Desm. & Rob.) West	Beech leaf spot
Gloeosporium inconspicuum Cav.	Elm anthracnose
Gloeosporium limetticolum Clausen	Lime anthracnose and withertip
Gloeosporium mezerei Cke.	Daphne leaf spot
Glomerella cingulata (Ston.) Spauld. & Schrenk	Bitter rot
Glomerella cingulata (Ston.) Spauld. & Schrenk var. vaccinii Shear	Cranberry bitter rot
Gnomonia caryae Wolf	Hickory anthracnose
Gnomonia caryae Wolf var. pecanae Cole	Pecan liver spot
Gnomonia platani (Sacc. & Speg.) Kleb.	Sycamore anthracnose
Gnomonia quercina Kleb.	Oak anthracnose
Gnomonia tiliae Kleb.	Linden anthracnose
Gnomonia ulmea (Schw. ex Fr.) Thuem.	Elm black spot
Graphiola phoenicis Pat.	Palm false smut
Guignardia aesculi (Pk.) V. B. Stewart	Buckeye leaf blotch Horsechestnut leaf blotch
Guignardia bidwellii (Ell.) Viala & Ravaz	Grape black rot
Guignardia vaccinii Shear	Cranberry early rot or blast
Gymnoconia peckiana (Howe) Trott.	Blackberry orange rust
Gymnosporangium clavipes Cke. & Pk.	Quince rust
Gymnosporangium cupressi Long & Goodding	Cypress rust
Gymnosporangium globosum Farl.	Hawthorn rust
Gymnosporangium juniperi-virginianae Schw.	Cedar-apple rust
Gymnosporangium libocedri (P. Henn.) Kern	Incense cedar rust
Helicobasidium purpureum Pat.	Violet root rot

Helminthosporium heveae Petch	Hevea birdseye spot
Helminthosporium papulosum Berg	Apple black pox
Helminthosporium torulosum (Syd.) Ashby	Banana black-tip
Herpobasidium deformans Gould	Honeysuckle leaf blight
Herpotrichia nigra Hartig	Coniferous brown felt blight
Heterodera marioni (Cornu) Goodey (See Meloidogyne spp.)	Root-knot nematode
Hypodermella abietis-concoloris (Mayr) Dearn.	Fir needle blight
Hypodermella laricis Tub.	Larch needle and shoot blight
Hydnum abietis Hubert	Fir brown pocket rot
Kuehneola malvicola (Speg.) Arth.	Hibiscus rust
Kuehneola uredinis (Lk.) Arth.	Rubus yellow-rust
Kunkelia nitens (Schw.) Arth.	Blackberry short-cycle orange rust
Leptosphaeria coniothyrium (Fckl.) Sacc.	Rose cane canker
Leptothyriella liquidambaris Tehon & Stout	Sweetgum red leaf spot
Leptothyrium pomi (Mont. & Fr.) Sacc.	Apple fly speck
Linospora gleditsiae Miller & Wolf	Honeylocust tar spot
Lophodermium juniperinum (Fr.) DeNot.	Juniper needle cast
Lophodermium piceae (Fckl.) Hoehn.	Spruce tar spot needle cast
Macrophoma fici Alm. & Cam.	Fig fruit canker
Macrophoma musae (Cke.) Berl. & Vogl.	Banana freckle
Marmor astri Holmes	Peach asteroid spot
Marmor caricae (Condit & Horne) Holmes	Fig mosaic
Marmor cerasi Zeller & Evans	Cherry mottle leaf
Marmor lineopictum Cation	Prunus line-pattern mosaic
Marmor mali Holmes	Apple mosaic
Marmor nerviclarens Zeller & Evans	Cherry vein-clearing mosaic
Marmor pallidolimbatus Zeller & Milbrath	Cherry banded-chlorosis
Marmor persicae Holmes	Peach mosaic
Marmor rosae Holmes	Rose mosaic
Marmor rubi Holmes	Red-raspberry mosaic
Marmor rubiginosum Reeves	Cherry rusty mottle

Marmor veneniferum Holmes	Rose streak
Melampsora abieti-capraearum Tub.	Fir-willow rust
Melampsora abietis-canadensis Ludwig	Canadian hemlock-poplar rust
Melampsora bigelowii Thuem.	Larch-willow rust
Melampsorella cerastii (Pers.) Schroet.	Yellow witches-broom
Melampsoridium alni (Thuem.) Diet.	Alder rust
Melampsoridium betulinum (Pers.) Kleb.	Birch leaf rust
Melanconis juglandis (Ell. & Ev.) Graves	Walnut canker and dieback
Meloidogyne spp. (Syn. Heterodera marioni (Cornu) Goodey)	Root-knot nematodes
Microsphaera alni DC. ex Wint. var. vaccinii (Schw.) Salm.	Vaccinium powdery mildew
Microsphaera grossulariae [Wallr.] Lév.	European powdery mildew
Microstroma juglandis (Bereng.) Sacc.	Walnut downy spot
Monilinia azaleae Honey	Azalea twig blight
Monilinia fructicola (Wint.) Honey	Brown rot of stone fruits Peach brown rot
Monilinia laxa (Aderh. & Ruhl.) Honey	European brown rot
Monilinia oxycocci (Wor.) Honey	Cranberry hard rot
Morenoella quercina (Ell. & Mart.) Theiss.	Oak purple blotch
Morsus suffodiens Holmes	Pierce's disease of grape
Morsus ulmi Holmes	Elm phloem-necrosis
Mycosphaerella caryigena Demaree & Cole	Pecan downy spot
Mycosphaerella musae (Speg.) Syd.	Banana Sigatoka disease
Mycosphaerella persica Higgins & Wolf	Peach frosty mildew
Nanus holodisci Holmes	Ocean-spray witches'-broom
Nanus loganobacci Holmes	Loganberry dwarf
Nanus mirabilis Holmes	Phony peach
Nanus orientalis Holmes	Raspberry streak
Nanus pruni Holmes	Prune dwarf
Nectria cinnabarina Tode ex Fr.	Coral spot
Nectria coccinea var. faginata Lohman, Watson & Ayers	Nectria beech bark canker

Nectria galligena Bres.	European Nectria canker
Neofabraea malicortis Jacks.	Northwestern apple anthracnose
Neofabraea perennans Kienholz	Perennial canker
Nummularia discreta (Schw.) Tul.	Apple blister canker
Oidium euonymi-japonici (Arcang.) Sacc.	Euonymus powdery mildew
Omphalia flavida (Cke.) Maubl. & Rangel	Coffee viruela or iron spot
Oospora citri aurantii (Ferr.) Sacc. & Syd.	Citrus sour rot
Ovularia maclurae Ell. & Langl.	Osage-orange cottony leaf spot
Ovulinia azaleae Weiss	Azalea flower blight Azalea flower spot
Pellicularia filamentosa (Pat.) Rogers	Web blight
Pellicularia koleroga Cke.	Thread blight
Peridermium ephedrae Cke.	Ephedra rust witches'-broom
Peronospora rubi Rabh.	Rubus downy mildew
Pestalotia funerea Desm.	Tip blight of conifers
Pestalotia macrotricha Kleb.	Rhododendron gray blight
Pezicula carpinea (Pers. ex Fr.) Tul.	Hornbeam branch and trunk canker
Phacidium infestans Karst.	Snow blight of conifer seedlings
Phleospora asiminae Ell. & Morgan	Common papaw leaf blotch
Pholiota adiposa Fr.	Brown mottled heart rot
Phoma abietina Hartig	Fir twig canker
Phoma cydoniae Sacc. & Schulz.	Quince fruit pale rot
Phoma persicae Sacc.	Peach stem canker
Phomopsis gardeniae Hansen & Barrett	Gardenia canker and stem gall
Phomopsis juniperovora Hahn	Cedar blight Juniper nursery blight
Phomopsis kalmiae Enlows	Mountain-laurel leaf blotch
Phoradendron californicum Nutt.	California mistletoe
Phoradendron coloradense Trel.	Mesquite mistletoe
Phoradendron densum Torr.	Dense mistletoe
Phoradendron engelmanni Trel.	Texas mistletoe
Phoradendron flavescens (Pursh) Nutt.	Eastern mistletoe

Phoradendron juniperinum Engelm.	Juniper mistletoe
Phoradendron juniperinum var. libocedri Engelm.	Incense cedar mistletoe
Phoradendron ligatum Trel.	Constricted mistletoe
Phoradendron longispicum Trel.	Long-spiked mistletoe
Phoradendron macrophyllum Cock.	Arizona mistletoe
Phoradendron pauciflorum Torr.	Fir mistletoe
Phoradendron villosum Nutt.	Hairy mistletoe
Phragmodothella ribesia (Pers. ex Fr.) Petr.	Currant black pustule
Phyllactinia corylea Pers. ex Karst.	Tree powdery mildew
Phyllosticta cotoneastri Allesch.	Cotoneaster leaf spot
Phyllosticta magnoliae Sacc.	Magnolia large leaf spot
Phyllosticta prunicola Sacc.	Apple scurfy bark
Phyllosticta solitaria Ell. & Ev.	Apple blotch
Phymatotrichum omnivorum (Shear) Dug.	Texas root rot Ozonium root rot Cotton root rot Omnivorous root rot
Physalospora miyabeana Fukushi	Willow black canker
Physalospora obtusa (Schw.) Cke.	Apple frogeye leaf spot
Physopella fici (Cast.) Arth.	Fig rust
Phytophthora cactorum (Leb. & Cohn) Schroet.	Bleeding canker Dogwood crown canker Lilac shoot blight
Phytophthora citrophthora (R.E. & E.H.Sm.) Leonian	Citrus brown rot and gummosis
Phytophthora palmivora Butl.	Cacao canker and pod rot Palm bud rot
Plasmopara ribicola Schroet.	Ribes downy mildew
Plasmopara viburni Pk.	Viburnum downy mildew
Plasmopara viticola (Berk. & Curt.) Berl. & de Toni	Grape downy mildew
Pleurotus ostreatus Fr.	White flaky sapwood rot
Pleurotus ulmarius Fr.	Elm brown sapwood rot
Podosphaera oxyacanthae (DC) DBy.	Cherry powdery mildew
Polyporus abietinus Dicks. ex Fr.	Fir pitted sapwood rot

Polyporus anceps Pk.	Red ray rot
Polyporus balsameus Pk.	Balsam fir butt rot
Polyporus betulinus Bull. ex Fr.	Birch powdery brown rot
Polyporus catalpae Schrenk	Catalpa brown butt rot
Polyporus dryadeus Pers. ex Fr.	Oak white root rot
Polyporus hispidus Bull. ex Fr.	White spongy heart rot
Polyporus pargamenus Fr.	White pocket rot
Polyporus robiniophilus (Murr.) Lloyd	Black locust white spongy heart rot
Polyporus schweinitzii Fr.	Red-brown butt rot
Polyporus sulphureus Bull ex Fr.	Brown-checked rot
Poria sequoiae Bonar	Redwood brown pocket heart rot
Poria subacida (Pk.) Sacc.	Feather rot
Pratylenchus spp.	Meadow nematodes
Pseudomonas aceris (Ark.) Starr & Burkh.	Maple bacterial leaf spot
Pseudomonas aleuritidis (McCul. & Demaree) Stapp	Aleurites bacterial leaf spot
Pseudomonas berberidis (Thornberry & H. W. Anders.) Stapp	Barberry bacterial leaf spot
Pseudomonas colurnae (Thornberry & H. W. Anders.) Burkh.	Turkish hazelnut bacterial leaf spot
Pseudomonas gardeniae Burkh. & Pirone	Gardenia bacterial leaf spot
Pseudomonas mori Boyer & F. Lambert	Mulberry bacterial spot-blight
Pseudomonas papulans Rose	Apple bacterial blister rust
Pseudomonas savastanoi (E. F. Sm.) F. L. Stevens	Olive bacterial knot
Pseudomonas savastanoi var. fraxini Brown	Ash bacterial canker
Pseudomonas syringae Van Hall	Citrus blast and black pit Lilac bacterial blight
Pseudomonas tonelliana (Ferr.) Burkh.	Oleander bacterial knot
Pseudomonas viburni (Thornberry & H. W. Anders.) Stapp	Viburnum bacterial leaf spot
Pseudoperonospora celtidis (Waite) G. W. Wils.	Hackberry downy mildew
Pseudopeziza ribis Kleb.	Currant anthracnose
Puccinia graminis Pers.	Stem rust

Puccinia malvacearum Mont.	Hollyhock rust
Puccinia peridermiospora (Ell. & Tracy) Arth.	Ash rust
Puccinia psidii Wint.	Common guava rust
Pucciniastrum americanum (Farl.) Arth.	Raspberry late leaf rust
Pucciniastrum ericae (Naumann) Cummins	Heath rust
Pucciniastrum goeppertianum (Kuehn) Kleb.	Fir-huckleberry rust
Pucciniastrum hydrangeae (Berk. & Curt.) Arth.	Hydrangea rust
Pucciniastrum pustulatum (Pers.) Diet.	Fir-fireweed rust
Rhabdocline pseudotsugae Syd.	Douglas-fir needle blight
Rhizina inflata (Schaeff.) Sacc. (= R. undulata Fr.)	Rhizina root rot
Rhytisma acerinum Pers. ex Fr.	Maple tar spot
Rhytisma andromedae Pers. ex Fr.	Andromeda tar spot
Rhytisma arbuti Phill.	Arbutus tar spot
Rhytisma punctatum Pers. ex Fr.	Black-speckled leaf spot
Rhytisma salicinum Pers. ex Fr.	Willow tar spot
Rhytisma vaccinii Schw. ex Fr.	Vaccinium tar spot
Rimocortius kwanzani Milbrath & Zeller	Flowering-cherry rough bark
Rimocortius psorosis (Fawcett) Holmes	Citrus psorosis
Rimocortius pyri Holmes	Pear stony pit
Rosellinia caryae Bonar	Hickory twig canker
Scleroderris abieticola Zeller & Goodding	Fir twig and branch canker
Sclerotinia camelliae Hara	Camellia flower blight
Sclerotium rolfsii Sacc.	Southern wilt
Septobasidium sabalis Couch	Palmetto brown felt
Septoria azaleae Vogl.	Azalea angular leaf spot
Septoria caryae Ell. & Ev.	Hickory leaf blight
Sphaceloma lippiae Baines & Cummins	Lippia spot anthracnose
Sphaceloma perseae Jenkins	Avocado scab
Sphaceloma poinsettiae Jenkins & Ruehle	Poinsettia spot anthracnose
Sphaceloma rosarum (Pass.) Jenkins	Rose spot anthracnose
Sphaceloma symphoricarpi Barrus & Horsfall	Snowberry spot anthracnose, scab

Sphaeropsis ulmicola Ell. & Ev.	Sphaeropsis canker of elm
Sphaerotheca humuli (DC) Burr.	Rose powdery mildew
Sphaerotheca lanestris Harkn.	Oak brown mildew
Sphaerotheca mors-uvae (Schw.) Berk. & Curt.	American powdery mildew
Sphaerulina taxi (Cke.) Mass.	Yew needle blight
Stemphylium citri Patterson & Charles	Citrus fruit end rot
Stereum fasciatum Schw.	Brown crumbly heart rot
Stereum purpureum Pers. ex Fr.	Silver leaf
Stereum sanguinolentum Alb. & Schw. ex Fr.	Red heart rot
Strumella coryneoidea Sacc. & Wint.	Strumella canker of chestnut and oak
Synchytrium vaccinii Thomas	Red leaf gall
Systremma acicola (Dearn.) Wolf & Barbour	Pine brown spot of needles
Taphrina aesculi (Patterson) Gies.	California buckeye witches-broom
Taphrina amelanchieri Mix	Amelanchier witches'-broom
Taphrina americana Mix	Birch witches'-broom
Taphrina australis (Atk.) Gies.	American hornbeam leaf curl
Taphrina bartholomaei Mix	Western maple leaf blister
Taphrina caerulescens (Mont. & Desm.) Tul.	Oak leaf blister
Taphrina carnea Johans.	Birch red leaf blister
Taphrina castanopsidis (Ell. & Ev.) Jenkins	California chinquapin leaf blister
Taphrina cerasi (Fckl.) Sadeb.	Cherry witches'-broom
Taphrina coryli Nishida	Hazelnut leaf blister
Taphrina dearnessii Jenkins	Maple black leaf blister
Taphrina deformans (Berk.) Tul.	Peach leaf curl
Taphrina flava Farl.	Birch yellow leaf blister
Taphrina populina Fr.	Poplar yellow leaf blister
Taphrina pruni (Fckl.) Tul.	Plum pockets
Taphrina saccharī Jenkins	Maple brown leaf blister
Taphrina ulmi (Fckl.) Johans.	Elm leaf blister
Taphrina virginica Sadeb.	Eastern hop hornbeam leaf curl
Thelephora terrestris Ehrh. ex Fr.	Smothering of seedlings
Thyronectria berolinensis (Sacc.) Seaver	Ribes cane knot

Torula ligniperda (Willk.) Sacc.	Pink wood stain
Trabutia erythrospora (Berk. & Curt.) Theiss. & Syd.	Oak black spot
Tranzschelia pruni-spinosae (Pers.) Diet.	Stone fruit rust
Tylenchulus semipenetrans Cobb	Citrus nematode
Uncinula clintonii Pk.	Linden powdery mildew
Uncinula flexuosa Pk.	Horse-chestnut powdery mildew
Uncinula necator (Schw.) Burr.	Grape powdery mildew
Uncinula prosopodis Speg.	Mesquite powdery mildew
Uncinula salicis DC. ex Wint.	Willow powdery mildew
Ustulina vulgaris Tul.	Ustulina butt rot
Venturia inaequalis (Cke.) Wint.	Apple scab
Venturia pirina Aderh.	Pear scab
Verticillium albo-atrum Reinke & Berth.	Verticillium wilt
Xanthomonas citri (Hasse) Dowson	Citrus canker
Xanthomonas corylina P. W. Miller et al.	Filbert bacterial blight
Xanthomonas juglandis (Pierce) Dowson	Walnut bacterial blight
Xanthomonas malvacearum (E. F. Sm.) Dowson	Bacterial angular leaf spot
Xanthomonas pruni (E. F. Sm.) Dowson	Bacterial blight of stone fruits
Xylaria mali Fromme	Apple black root rot

PARTIAL BIBLIOGRAPHY

Anderson, P. J., R. J. Haskell, W. C. Muenscher, D. J. Weld, J. I. Wood, and G. H. Martin. Check list of diseases of economic plants in the United States. U. S. Dept. Agr. Bul. 1366. 1926.

Boyce, John Shaw. Forest Pathology. Ed. 2. 1948.

Breed, R. S., E. D. G. Murray, and A. P. Hitchens. Bergey's manual of determinative bacteriology. Ed. 6. 1948.

British Mycological Society. List of common British plant diseases. Ed. 3. 1944.

Chester, K. S. The nature and prevention of plant diseases. 1942.

Crowell, I. H., and E. Lavallée. Check list of diseases of economic plants in Canada. 1942.

Dodge, B. O., and H. W. Rickett. Diseases and pests of ornamental plants. Rev. Edition. 1948.

Gill, L. S. Arceuthobium in the United States. Connecticut Acad. Arts and Sci. Trans. 32, p. 111-245. 1935.

Fawcett, H. S., and H. A. Lee. Citrus diseases and their control. Ed. 1. 1926.

Felt, E. P., and W. H. Rankin. Insects and diseases of ornamental trees and shrubs. 1932.

Heald, F. H. Manual of plant diseases. Ed. 2. 1933.

Hesler, L. R., and H. H. Whetzel. Manual of fruit diseases. 1924.

Hildebrand, E. M., G. H. Berkeley, and D. Cation. Handbook of virus diseases of stone fruits in North America. Michigan Agr. Exp. Sta. Misc. Publ. (unnumbered) 1942.

Holmes, F. O. Handbook of phytopathogenic viruses. 1939.

Kelsey, H. P., and W. A. Dayton. Standardized plant names. Ed. 2. 1942.

Lowe, J. L. The Polyporaceae of New York State (except Poria). New York State Col. Forestry, Syracuse Univ. Tech. Bul. 60, Vol. XV, No. 3. 1942.

Marshall, R. P., and A. M. Waterman. Common diseases of important shade trees. U. S. Dept. Agr. Farmers' Bul. 1987. 1948.

Mix, A. J. A monograph of the genus Taphrina. Kansas Univ. Sci. Bul. Vol. 33, Part 1, No. 1, p. 3-167. 1949.

Muesebeck, C. F. W. Common names of insects approved by the American Association of Economic Entomologists. Jour. Econ. Ent. 43: 117-138. 1950.

Shear, C. L. Report of the Seventh annual meeting of the American Phytopathological Society - Committee on Common Names of Plant Diseases. Phytopathology 6: 211-212. 1916.

Stevens, F. L. Plant disease fungi. 1925.

Trelease, W. The genus Phoradendron. Published by the University, Urbana, Illinois. 1916.

Weiss, F. Index of plant disease in the United States. U. S. Dept. Agr. Plant Disease Survey Special Publication. Parts I, II, III. 1950.

_____. Check list revision, diseases of economic plants of the United States. Plant Dis. Reptr. 24 (7) through 27 (1). 1940-1943.

Westcott, C. The plant doctor. Ed. 3. 1950.

_____. Plant disease handbook. 1950.

DIVISION OF PLANT QUARANTINES, BUREAU OF ENTOMOLOGY AND PLANT QUARANTINE, AGRICULTURAL RESEARCH ADMINISTRATION, WASHINGTON, D. C.

As already noted by the compiler, this list is limited to diseases of woody plants and has been prepared of course with the special needs of the plant quarantine service in mind. It is not intended as an official list, but rather to point the way to further work looking toward the standardization of common names of plant diseases. The list is published by the Plant Disease Survey in the hope that it will stimulate further contributions along the same line so that eventually data may be available for a list that can be officially adopted. P.R.M.

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 208

THE PLANT DISEASE WARNING SERVICE IN 1951

Supplement 208 December 15, 1951

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

THE PLANT DISEASE WARNING SERVICE IN 1951

Paul R. Miller and Muriel O'Brien

Plant Disease Reporter
Supplement 208 December 15, 1951

INTRODUCTION

Outstanding features of the 1951 season were a destructive epidemic of blue mold in Connecticut shade tobacco fields, and continuance of the northwestward advance of late blight as noted last year. In general, of the diseases handled under the Crop Plant Disease Forecasting Program, namely, late blight of potato and tomato (Phytophthora infestans (Mont.) D By.), tobacco blue mold (Peronospora tabacina Adam), and cucurbit downy mildew (Pseudoperonospora cubensis (Berk. & Curt.) Rostow.), late blight on both hosts was the most widespread and active in 1951. Cucurbit downy mildew and tobacco blue mold were negligible in incidence and spread, except for the Connecticut field outbreak of blue mold.

In the case of late blight, factors in its incidence and spread were its non-appearance in commercial greenwrap acreages planted with home-grown rather than out-of-state plants (Mississippi), the operation of good control programs when necessary, and the warm, dry weather in mid- to late season over the eastern portion of the United States. On the other hand, its widespread activity in the eastern Provinces of Canada was probably owing to the favorable weather conditions for blight development prevailing throughout that area this summer.

Concerning tobacco blue mold, absence of overwintering inoculum in certain areas prevented development of the disease. Adequate amounts of spray and dust materials were readily available and were utilized. Despite the above-normal January temperatures, light rainfall and average temperatures in the following spring months in tobacco-growing areas, together with control measures, held blue mold at a low level.

Cucurbit downy mildew was active this year in regions where it is endemic on squash and cucumbers. Cucurbit-growing areas of Texas escaped the disease this year owing to hot, dry weather conditions. A feature of downy mildew occurrence this year was the absence of its usual progressive northward development. The disease was found in one place in Virginia, was not noticed in Maryland or Delaware, but appeared in Pennsylvania.

PHYTOPHTHORA INFESTANS ON TOMATO:

Tomato late blight this year continued its northwestward movement across the upper tier of our States (Fig. 1). Although widespread acreages were affected, reduction in yields was low. Sources of infection included potato dump piles, nearby potato plants, airborne spores, southern transplants, and greenhouse plants. Along with the operation of good control programs, the hot, dry weather in mid- to late season helped check the further development of blight.

Fungicides used as sprays and dusts were effective in controlling the disease, especially when schedules were not interrupted by cold spells and where sufficient coverage was obtained. Losses in some cases would have been greater than in 1950 except for the fact that many canners and market gardeners used fungicides, the small growers showing interest in using large sprayers for better coverage. As listed in Table 1, products used included Dithane, Parzate, fixed coppers, Bordeaux mixture, nabam plus zinc sulfate, tribasic copper sulfate, zineb, Copper A, and carbamates.

PHYTOPHTHORA INFESTANS ON POTATO:

Figure 2 shows the wide distribution of potato late blight throughout the United States and the eastern Provinces of Canada. Blight on potatoes also continued its northwestward advance as noted in last year's summary. Estimated State averages of reduction in yield in infected acreages varied from 2 percent to as high as 50 percent tuber loss. Sources of inoculum included airborne sporangia, infected seed from the previous year, and nearby cull piles.

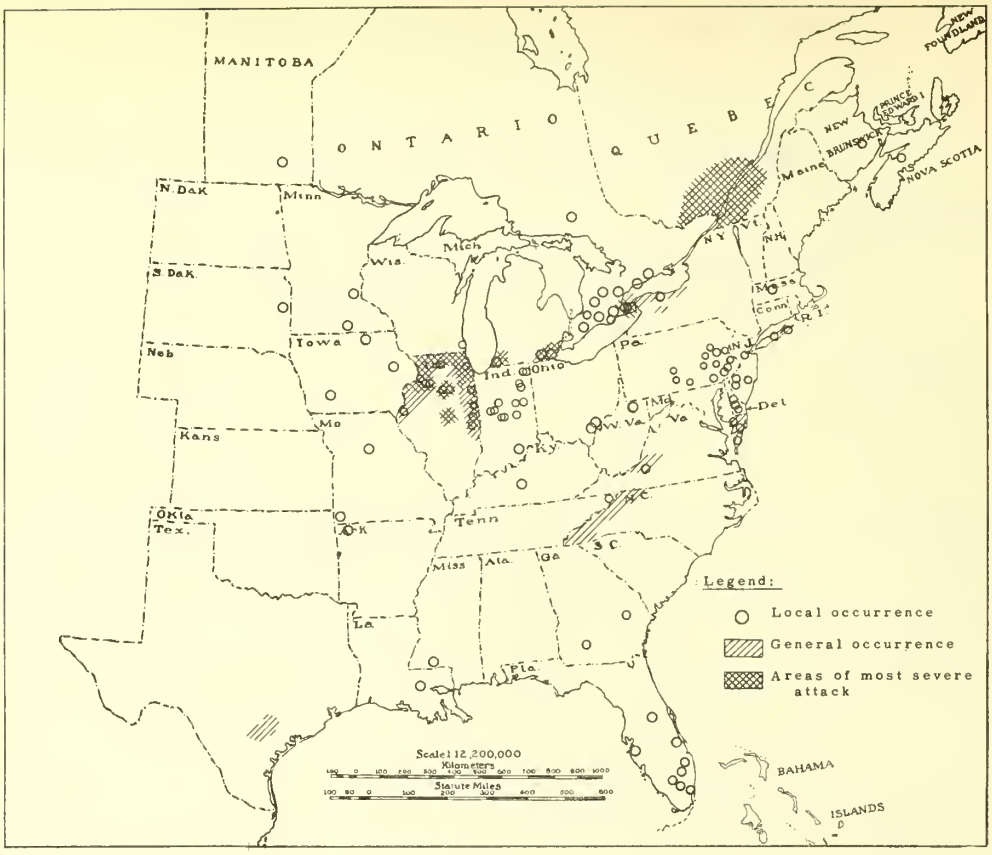


FIG. 1. DISTRIBUTION AND IMPORTANCE OF TOMATO LATE BLIGHT IN 1951.

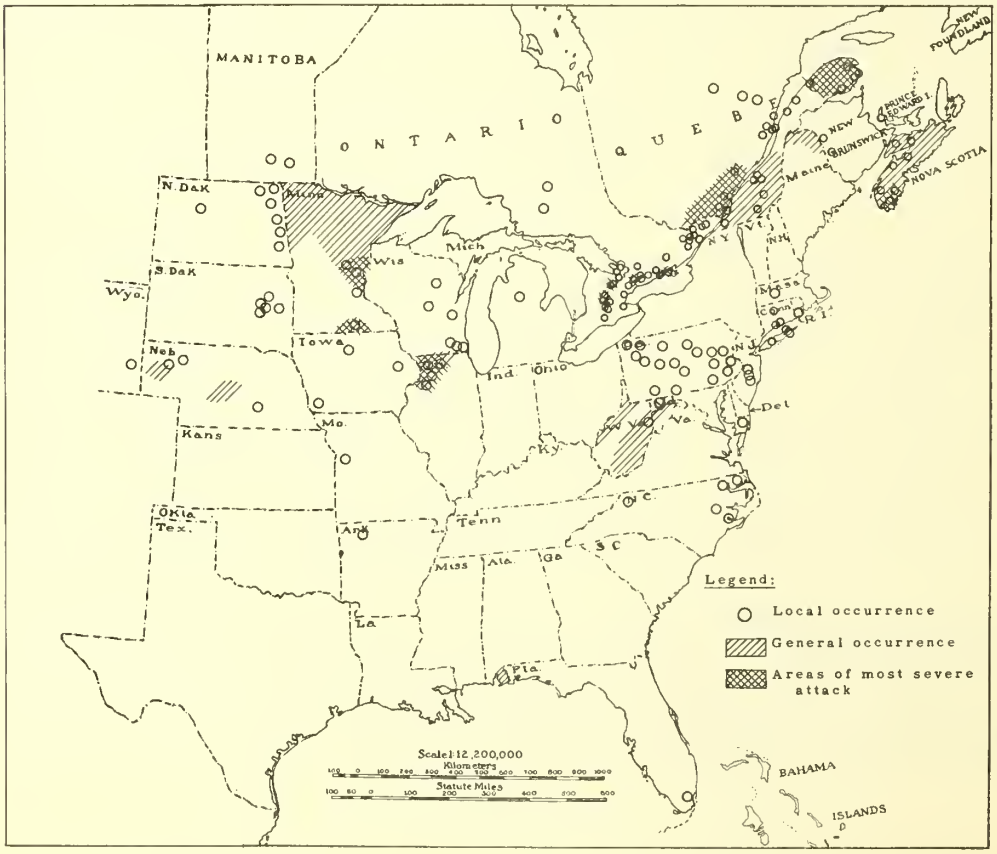


FIG. 2. DISTRIBUTION AND IMPORTANCE OF POTATO LATE BLIGHT IN 1951.

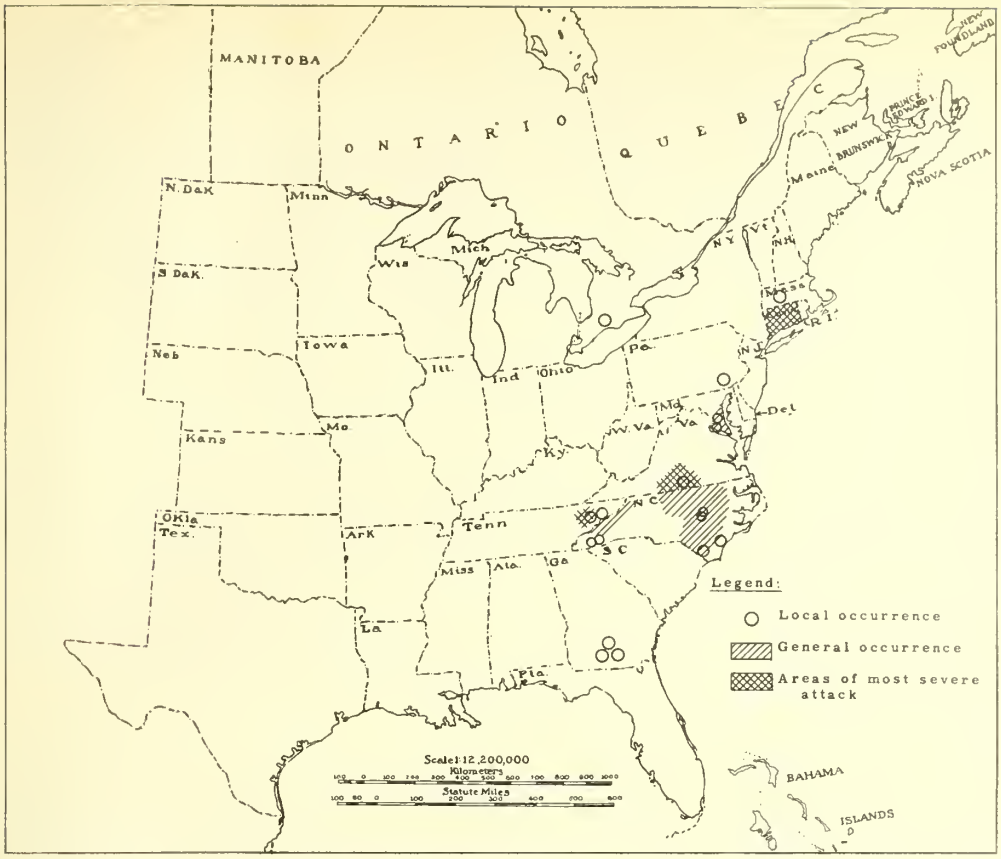


FIG. 3. DISTRIBUTION AND IMPORTANCE OF TOBACCO BLUE MOLD IN 1951.

FIG. 4. DISTRIBUTION AND IMPORTANCE OF CUCURBIT DOWNY MILDEW IN 1951.

Lack of appreciable spread in many instances was attributed to good disease control programs. The dry, warm weather in mid- to late season experienced in some of the potato-growing areas also served to check the advance of blight.

Fungicides used in the control of potato blight, as listed in Table 2, included Dithane, Parzate, fixed coppers, nabam plus zinc sulfate, Bordeaux mixture, and zineb.

PERONOSPORA TABACINA ON TOBACCO:

The distribution of tobacco blue mold in 1951 is shown in Fig. 3. The disease on the whole was very light this year. Average reduction in yield of acreages affected ranged from 0 to 3 percent.

The one exception to the generally light attack of blue mold this year was in Connecticut where shade growers sustained a loss of 10 percent of the value of the crop, or about $2,000,000, from a devastating outbreak in shade tobacco fields. Damage to seedbeds had been less than in any other year since the disease was first found in the State. This relative freedom from disease undoubtedly led growers to relax control efforts. Appearance of blue mold in the seedbeds after plants had been set out was not looked upon as particularly dangerous since spread of the disease to the field is unusual. However, this extensive source of inoculum, plus a cool, wet, early summer, evidently afforded ideal conditions for initiation and development of an unprecedented field epidemic. Control programs, when started, came too late and too little material was used to be effective.

Fungicides listed in Table 3 indicate a preference for Fermate. Along with Fermate, Dithane Z-78, Parzate, and zineb also afforded good to excellent control. Unfavorable weather conditions for blue mold development and the availability of plenty of plants also helped to reduce loss.

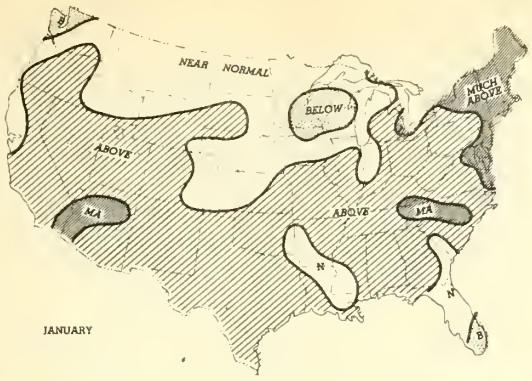
PSEUDOPERONOSPORA CUBENSIS ON CUCURBITS:

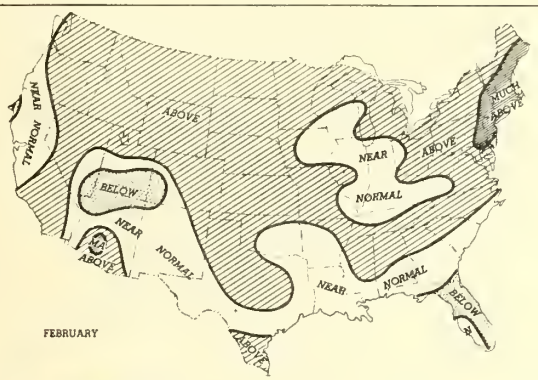
Cucurbit downy mildew occurred along the Atlantic Coast seaboard as far north as Pennsylvania, where its occurrence is noteworthy in view of the fact that the disease failed to cause much damage in North Carolina where it was generally distributed, and did not occur in Maryland and Delaware. Losses were at a minimum and the disease did not figure prominently in the disease picture this year. Weather conditions in some instances were not favorable for disease development; or it appeared late, causing only slight injury. Fig. 4.

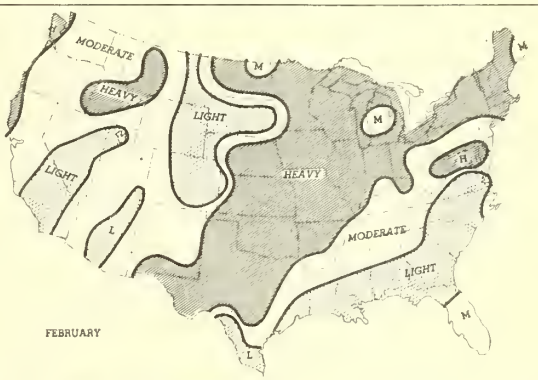
Fungicides used in controlling cucurbit downy mildew, Table 4, included zineb, nabam plus zinc sulfate, fixed copper, and ziram. Only a small percentage of growers use control measures but where nabam plus zinc sulfate and zineb were used as sprays, excellent results were obtained.

WEATHER

Maps, showing approximate observed temperatures and precipitation for the months of January through September, are shown in Figs. 5a, b, and c.







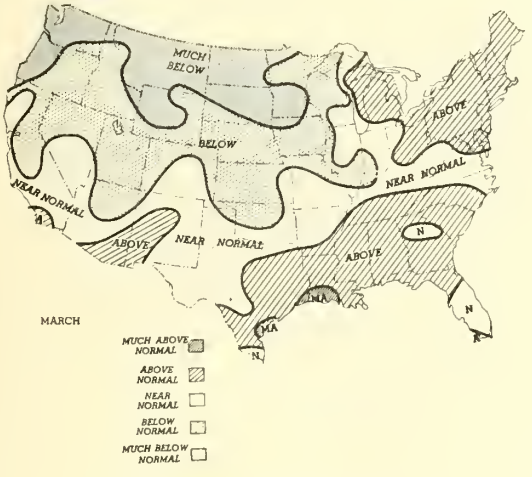
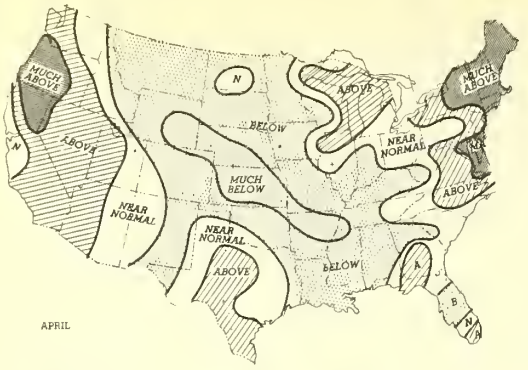
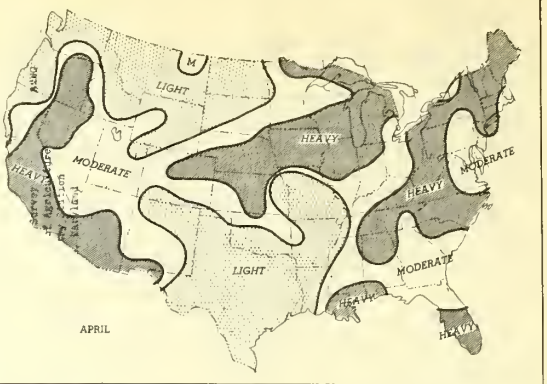
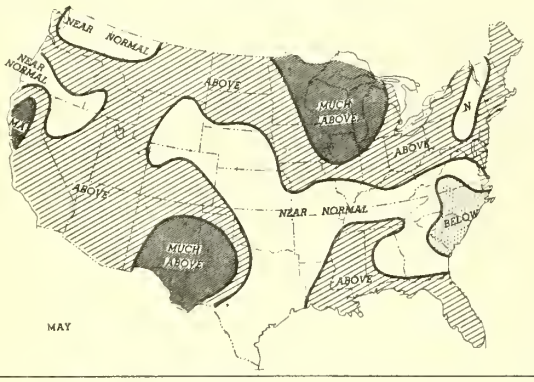


Fig. 5a. APPROXIMATE OBSERVED TEMPERATURE
JANUARY through MARCH, 1951.

APPROXIMATE OBSERVED PRECIPITATION
JANUARY through MARCH, 1951







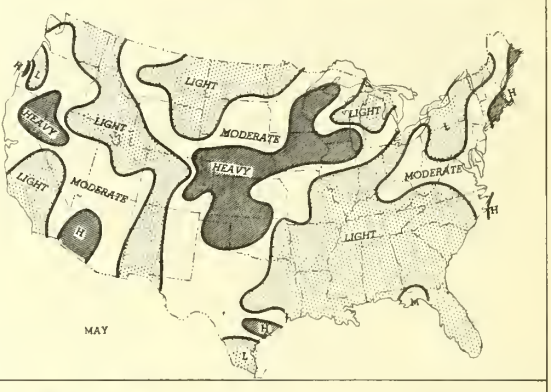


Fig. 5b. APPROXIMATE OBSERVED TEMPERATURE - APRIL through JUNE, 1951

APPROXIMATE OBSERVED PRECIPITATION - APRIL through JUNE, 1951

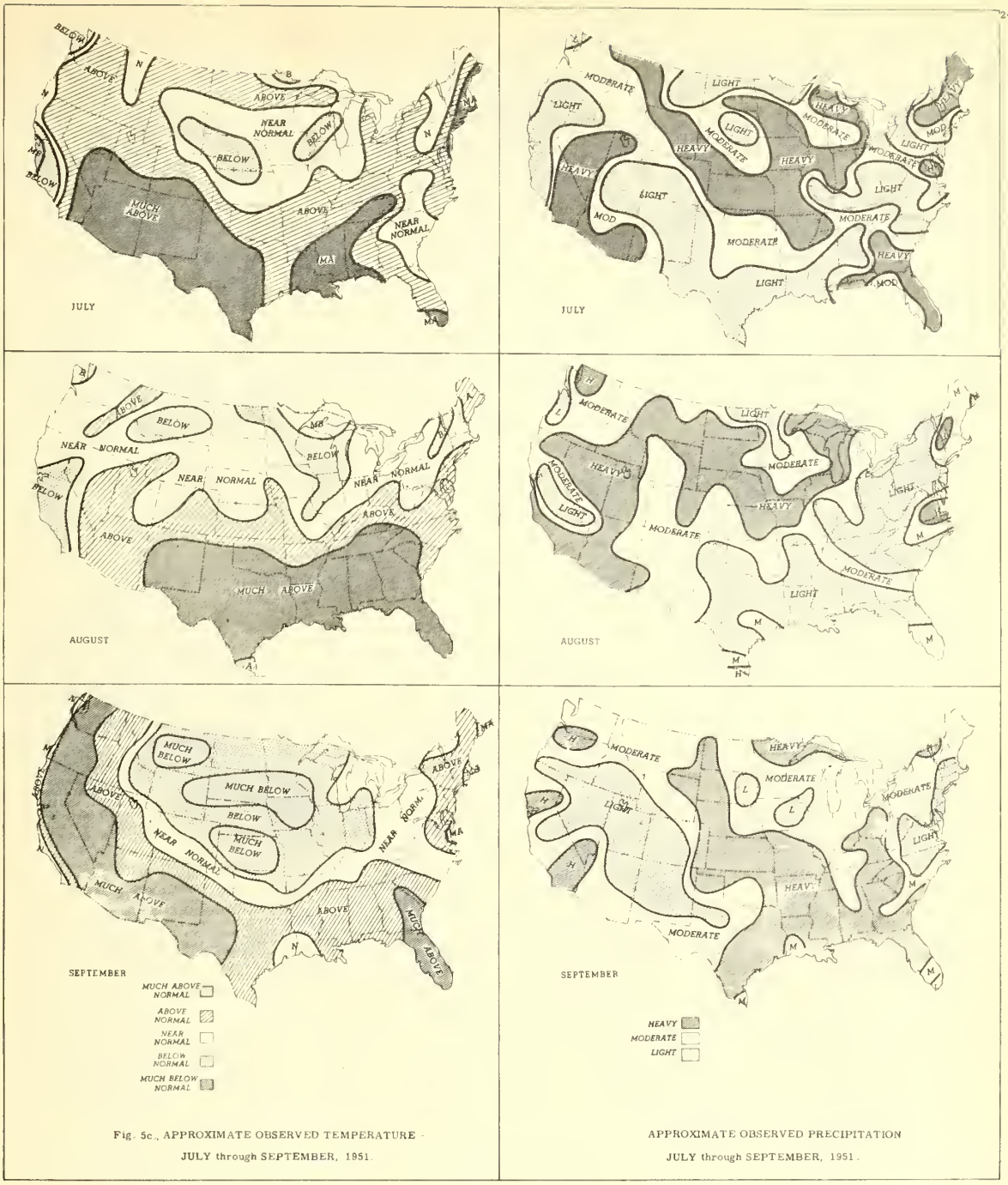


Fig. 5c., APPROXIMATE OBSERVED TEMPERATURE - JULY through SEPTEMBER, 1951.

APPROXIMATE OBSERVED PRECIPITATION JULY through SEPTEMBER, 1951.

Table 1. CONTROL OF LATE BLIGHT ON TOMATO: Materials used as Sprays and Dusts and their effectiveness, 1951.

State or Province	Materials	Concentration	Percent growers using	Results	Remarks
SPRAYS:					
Ala.	Nabam	2 qts. - 3/4 lb. $ZnSO_4$-100	1		No late blight present.
	Zineb concentrate	2 lbs.-100 gals.	.05		
Conn.	COCS	4-100	Unknown	All gave good control	Owing to press, radio, and personal warnings, most growers applied control earlier and more often.
	Tribasic	"	"		
	Copper A	"	"		
	Bordeaux	"	"		
Del.	Zineb	2 qts.-1-100 or 2-100	80	Good all season	The disease was of little consequence in the commercial canning crop. It damaged some late open-market fields.
	Tribasic	3-100	30	Good August application only	
	Bordeaux	6-3-100	30	Good August application only	
Fla. (Dade Co.)	Nabam + $ZnSO_4$	2-1-100	80	Good to excellent	Since blight appeared in seedbeds at transplanting time, it was in some cases carried to the field causing poor stands, necessity of replanting in a few cases. Thirteen cold spells interrupted spray schedules.
	Zineb	2-100	20	" " "	
Ill.	Dithane D-14 + $ZnSO_4$	2 qts.-1 lb.-100	70	Where fungicides were applied before late blight became too severe, all materials gave good to excellent control with the exception of Zerlate.	
	Zerlate	2 lbs.-100			
	Fixed coppers	4 lbs.-100			
	Parzate (dry)	2 lbs.-100			
	Dithane Z-78	2 lbs.-100			
	Bordeaux mixture	?			
Md.	Z-Z-T-T-T	2 lbs.& 4 lbs.-100 gal.	50	Good	Dry weather during mid- and late season retarded disease development.
	Dithane Z-78	2 lbs.-100 gal.	25	Good	
	Dithane D-14 + $ZnSO_4$	2 qts.-1 lb.-100 gal.	25	Good	
Mich.	Alternating copper and zerlate	Usual		Good	
	Tank mix copper and zerlate	"		Good	
	Dithane	"		Good in southeastern area; poor in southwestern	

Table 1. (Continued)

State or Province	Materials	Concentration	Percent growers using	Results	Remarks
SPRAYS: (Cont.)					
Miss.	Copper A Parzate Tribasic C.S. Dithane Z-78	4-100 2-100 4-100	85		Control measures used by growers in Copiah, Lincoln and Hinds Counties; greenwrap acreage very low, hence high percentage of growers using sprays and dusts.
N. H.	Neutral copper		Very few	Fair	No commercial acreage. Home gardeners suffered heavy losses; very few attempted to control the disease.
N. Y.	Zerlate - bordo Zineb	2, 8-4-100 2-1-100	70-80 10-15	Excellent Excellent in 1951	
N. C.	Fixed coppers Carbamates	2 lbs./100 2/100	15 5	Good Fair	
Pa.	Fixed copper Bordeaux Zineb	4 lbs. of 50% 6-3-100 8-4-100	Est. 70 15 5	Very good Excellent Good	To control other diseases most growers use ziram either in early sprays combined with fixed copper or in alternating sprays.
S. C.	Nabam	2-1-100	3	No disease	
Va.	Copper Zineb	2 lbs.-100 gal. 1 1/2 lbs. -100 gal.	10 1	Not enough late blight to judge efficacy of sprays.	Late blight started in eastern and southwestern Virginia but the weather conditions became unfavorable for blight which caused very little damage.
W. Va.	Zineb Bordeaux mixture Fixed coppers	2 1/2 lb.-100 8-8-100 2 lb. -100	10 10 10	Excellent Good Good	
Wis.	Copper A Carbamates	6 lb. -100 gal. 2-3 qts. -100 gal.	75 Very few	Poor ?	
DUSTS:					
Ala.	Zineb Tribasic copper	6% 6%	33 12		

Table 1. (Continued)

State or Province	Materials	Concentration	Percent growers using	Results	Remarks
DUSTS: (Cont.)					
Ark.	Tribasic copper sulfate	7% Cu.	Less than 1	No blight where used.	
	Dithane Z-78	6%	Less than 1	" " "	
Conn.	Fixed copper	8% Cu.	?	Fair control until late Sept. when dusting was stopped	
Del.	Tribasic		10	Fair	Disease was of little consequence in the commercial canning crop. Damaged some late open-market fields.
	Zineb		10	Fair	
Ill.	Fixed coppers	7% Cu.	6% where dusting service available	See Remarks under Sprays	
Md.	Ziram	10%		Fair	Dry weather during mid- and late season retarded disease development.
	Fixed copper	7%			
Mich.	Tribasic copper			Good	
Miss.	Copper A	12%			See Remarks under Sprays
	Parzate	"			
	Tribasic copper sulfate	"			
	Dithane Z-78	"			
N. C.	Fixed coppers	7% Metallic Cu.	75	Good	
	Carbamates	6% active	5	Fair	
N. H.	Neutral copper		Very few	Fair	No commercial acreage
N. Y.	Zerlate - copper	10% - 7%	5	Poor	
Pa.	Fixed copper	7%	10	Fairly good	
S. C.	Fixed copper	6-6 1/2% Cu.	50		State recommendations - copper with zineb or nabam as alternates.
Va.	Copper	6%	Few		Not enough late blight to judge effectiveness of control.

Table 1. (Continued)

State or Province	Materials	Concentration	Percent growers using	Results	Remarks
DUSTS: (Cont.)					
W. Va.	Zineb	6 1/2%	15	Excellent	
	Fixed coppers	6 - 7%	20	Good	
	Copper-lime	20-80	10	Good	
Wis.	Copper	7%	10	Poor	

Table 2. CONTROL OF LATE BLIGHT ON POTATO: Materials used as Sprays and Dusts and their effectiveness, 1951

State or Province	Materials	Concentration	Percent growers using	Results	Remarks
SPRAYS:					
Fla. (Dade Co.)	Nabam + Zinc sulfate	2-1-100	100	Excellent	Frequent cold "spells" interrupted regular spray.
Minn.	Dithane & Parzate coppers		95	Poor to good	Results depended on care in application and weather.
Nebr.	Dithane		2	Good	
	Parzate		1	"	
	Fixed copper		1	"	
N. H.	Neutral copper	6 lbs.-100 gal.	45	Good-	Home gardeners suffered heavy losses. Commercial growers did a fairly good job of controlling late blight. Kennebec acreage was fairly large and stood up well.
	Dithane, Parzate		5	Good	
Pa.	Zineb	2 qts. liquid + 1 lb. zinc sulfate	60	Good through season; not up to copper at end for storage rot	Ninety-nine percent+ of commercial growers use control measures.
	Bordeaux	8-4-100	40	Good in all stages	
	Fixed copper	4 lbs. of 50%	10	Good	
	Crag 658	2 lbs.	Few	Good	
S. C.	Nabam	2-1-100	1		No disease.
W. Va.	Zineb	2 1/2 lbs.-100	5	Excellent	
	Bordeaux mixture	8-8-100	10	Good	
	Fixed coppers	2 lbs.-100	5	Good	
Wis.	Carbamates	2 qts.-100-1 acre	?	Excellent to good	
	Copper (fixed)	4-6 lbs.-100-1 acre	?	Excellent to good	
	Bordeaux	8-10 lbs.5-6 lbs.-100 gals.	?	Excellent to good	
	Copper zinc chromate	2 lbs.-100-1 acre	?	Poor when 2 lbs. per 100 gal. used; 3-4 lbs. may give good control.	
Canada (Eastern Ontario)	Bordeaux mixture	4-2-100	83	Good	Late blight appeared early and spread somewhat slowly being severe in unsprayed and poorly sprayed fields, making conditions favorable for tuber infection. Blight appeared on potatoes one month earlier than tomatoes.
	Dithane	2-100	2	Poor to good	
	Fixed coppers	Various	3	Poor to good	

Table 2. (Continued)

State or Province	Materials	Concentration	Percent growers using	Results	Remarks
SPRAYS: (Cont.)					
(Prince Edward Island)	Bordeaux	80-4-80	65	Good to excellent	
	Dithane D-14	2 qts.-1 Zn-80	10	Good to excellent	
	Neutral coppers		10	Good	
DUSTS					
Minn.	Dithane, Parzate Fixed coppers		95	Poor to good Depending on care in application and weather.	
Nebr.	Dithane		1	Fair	
N. H.	Neutral copper	7% Cu.	45	Good	
Pa.	Copper	20-80%	Few		
S. C.	Fixed copper	6-6 1/2% Cu.	50		
	Zineb	4%	5		
W. Va.	Zineb	6 1/2%	15	Excellent	
	Fixed coppers	7%	20	Good	
	Copper-lime	20-80%	5	Good	
Wis.	Copper	7%			Very little used. About one-half dozen growers in Antigo area dusted with plane once or twice in addition to regular spray program. A few growers in southern and southeastern portion of State also used dust.
Canada (Eastern Ontario)	Fixed coppers	Various	2	Poor to good	
(Prince Edward Island)	Dithane Z-78		1	Poor	
	Coppers		2	Poor	

Table 3. CONTROL OF BLUE MOLD OF TOBACCO: Materials used as Sprays and Dusts and their effectiveness, 1951.

State or Province	Materials	Concentration	Percent growers using	Results	Remarks
SPRAYS:					
Md.	Fermate	2-100	50	Good	Hot, dry weather starting soon after initial infection stopped the disease and prevented serious losses.
N. C.	Fermate	4-100	27	Good	The attack of blue mold was later than usual; in the Border Belt most of the crop was set before blue mold became active. Elsewhere the disease became destructive just prior to and during the transplanting season on non-treated beds. Where the disease was severe, plants were not killed but transplanting was delayed.
	Dithane Z-78 or Parzate	3-100	3		
Tenn.	Ferbam	76% material, using 5 tablespoons/gallon	Est. 20	Good	
	Zineb	65% material, 2 1/2 tablespoons/gallon	Very few		
	Fermate	4 lbs.-100 gal.	40	Excellent	
	Dithane	3 lbs.-100 gal.	5	Good	
Va.	Ferbam	3-4 lb.-100 gal.	75	Excellent	Weather conditions mostly favorable for control of the disease; plenty of plants.
	Zineb	2 lbs.-100 gal.	Few	"	
DUSTS:					
Fla.	Ferbam	15.6%	40	Good	Excellence of results with zineb probably due to better coverage. Dusts applied 3 times a week using from 15 to 35 lbs. per acre depending on size of plants.
	Zineb	6.5%	40	Excellent	
Md.	Fermate	15%	50	Good	See Remarks under Sprays.
N. C.	Fermate	15%	30	Good	See Remarks under Sprays.
	Dithane Z-78 or Parzate	10%	5	Good	
Tenn.	Ferbam	10%	15	Good	
	Zineb	5%	15	Good	
	Fermate	10%	25	Excellent	
	Dithane	5%	5	Good	
Va.	Ferbam	10%	15	Excellent	Weather conditions mostly favorable for control of blue mold; plenty of plants.
	Zineb	6%	Few	"	

Table 4. CONTROL OF DOWNY MILDEW OF CUCURBITS: Materials used as Sprays and Dusts and their effectiveness, 1951.

State or Province	Materials	Concentration	Percent growers using	Results	Remarks
SPRAYS					
Fla.	Nabam + zinc sulfate	2-1-100		Excellent	Only a few farmers spray squash; with increased yields and good control, this number is increasing.
	Zineb	2-100		"	
S. C.	Nabam	2-1-100	5		Disease appeared after fungicide application stopped.
Va.	Copper	1 1/2 lb.-100	Few		Only a few growers used control measures. Weather conditions were not favorable for disease. Mildew appeared late and caused slight injury.
	Zineb	1 lb.-100	"		
DUSTS					
N. C.	Fixed copper (Tribasic)	5%	15	Poor to good	
	Carbamates	6%	2	Poor to fair	
S. C.	Fixed copper		38	Disease appeared late after dusting and spraying stopped and when harvest completed.	Recommendations: Copper (1st) zineb or nabam or ziram (2nd).
	Zineb		30		
	Ziram		2		
Va.	Copper	5%	Few		Only a few growers used control measures. Weather conditions were not favorable for disease. Mildew appeared late and caused slight injury.
	Zineb	6%	"		

THE PLANT DISEASE REPORTER

Issued By

THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AGRICULTURAL RESEARCH ADMINISTRATION

UNITED STATES DEPARTMENT OF AGRICULTURE

SUPPLEMENT 209

INDEX TO SUPPLEMENTS 199-208, 1951

Supplement 209 Issued March 15, 1952

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

PLANT DISEASE REPORTER SUPPLEMENT

Issued by

THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

Plant Industry Station Beltsville, Maryland

INDEX TO PLANT DISEASE REPORTER SUPPLEMENTS 199-208, 1951

Compiled by Nellie W. Nance

Plant Disease Reporter
Supplement 209

Index to Supplements
1951

LIST OF SUPPLEMENTS

Supplement 199. Wheat leaf rust studies at Saint Paul, Minnesota. pp. 3-17. March 30, 1951. Three lines of investigations are summarized, viz.: (1) The occurrence and distribution of physiologic races isolated from wheat leaf rust specimens, collected in the Upper Midwest area of the United States during the 25-year period, 1925-1949, inclusive; (2) the seedling reaction of more than a hundred varieties of wheat to different physiologic races of Puccinia rubigo-vera tritici, tested under greenhouse conditions; and (3) the comparative reaction of some six dozen wheat varieties, grown in field plots at University Farm during one or more of the 10 years from 1938 to 1947, inclusive. See author index below.

Supplement 200. Plant pathological investigation in the United States III. pp. 20-55. March 30, 1951. See its table of contents and author index below.

Supplement 201. A key to species of Helminthosporium reported on grasses in the United States. pp. 58-67. May 15, 1951. By E. S. Luttrell.

Supplement 202. Some new and important plant disease occurrences and developments in the United States in 1950. pp. 70-91. May 15, 1951. Compiled by Nellie W. Nance.

Supplement 203. Plant pathological investigation in the United States IV. pp. 94-107. June 15, 1951. Plant disease research and extension in Iowa. By W. F. Buchholtz and J. R. Wallin.

Supplement 204. Bibliography of soybean diseases. pp. 110-173. June 15, 1951. This bibliography covers approximately 500 titles published from 1882 to 1950, including a number on soybean diseases in the Orient, which are not easily accessible to western readers. By Lee Ling.

Supplement 205. Fungicidal and phytotoxic properties of 412 synthetic organic compounds. pp. 176-189. July 15, 1951. A previous publication discussed the fungicidal and phytotoxic properties of 506 synthetic organic chemicals. This paper gives the results of similar tests with 412 additional synthetic organic compounds. By M. C. Goldsworthy and S. I. Gertler.

Supplement 206. Plant pathological investigation in the United States V. Research in plant pathology and botany at Louisiana State University. pp. 193-201. September 15, 1951. By C. W. Edgerton.

Supplement 207. Common names of diseases of woody plants. pp. 205-235. September 15, 1951.

Supplement 208. The plant disease warning service in 1951. pp. 237-251. December 15, 1951.

Supplement 209. Index to Supplements 199 to 208. pp. 253-263. (Issued March 15, 1952).

AUTHOR INDEX

SUBJECT INDEX

ERRATA

CORRECTION FOR SUPPLEMENT 202

Our attention has been called to an error in Table 2, page 74 of Supplement 202, under DELPHINUM. Diplodinia delphinii was reported in Connecticut, New Jersey and New York as "A new Phoma disease of perennial delphinium." (Abstr.) Phytopath. 30:15. 1940, and not in 1950 as stated in this Supplement.

CORRECTION FOR SUPPLEMENT 147
(From PDR 35(11):511)

Reference to the occurrence of Sphaceloma sp. on soybean (Glycine max) collected in Franklin County, Pennsylvania, in the year 1943, and recorded in Plant Disease Reporter Supplement 147 on page 155, should be deleted. It was included in the summary concerned only through an oversight. The diseased specimen of soybean was insufficient to determine the identity of the involved pathogen and was not preserved. In 1943 Sphaceloma was not known on soybean, but it has since been found causing a destructive disease of this crop in Japan. (Jenkins, Anna E. Sphaceloma scab, a new disease of soybean discovered by plant pathologist in Japan. PDR 35: 110-111. 1951) -- L. J. Tyler, Department of Plant Pathology, Cornell University, Ithaca, New York.